SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS

A THESIS SUBMITTED TO

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT (PUNJAB)

IN THE PARTIAL FULFILLMENT

FOR THE AWARD OF DEGREE OF

MASTER OF SURGERY

IN

OTORHINOLARYNGOLOGY (ENT)

2023

Submitted By

DR. DEEKSHA CHAWLA

DEPARTMENT OF OTORHINOLARYNGOLOGY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA, PUNJAB

DECLARATION BY THE CANDIDATE

I hereby declare that the thesis entitled "**SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS**" submitted towards partial fulfillment of the requirements for the award of **MS in Otorhinolaryngology (ENT)** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Rohit Verma**, Professor, Department of ENT, **Dr. Manish Munjal,** Professor & Head, Department of ENT, **Dr. Siddharth Bhargava**, Associate Professor, Department of Paediatrics. No part of this thesis has formed the basis for the award of any degree previously.

_____

DR. DEEKSHA CHAWLA
Department of ENT,
(Otorhinolaryngology)
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

Place:

CERTIFICATE OF SUPERVISORS

This is to certify that the work incorporated in this thesis entitled "**SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS"** has been carried out by **Dr. Deeksha Chawla** under our direct supervision. The data incorporated in this thesis is original and genuine and has been carried out by the candidate herself.

Signature of Supervisor

Dr. Rohit Verma
Professor
Department of ENT
DMC&H, Ludhiana

Signature of Co-Supervisors

Dr. Manish Munjal
Professor & Head
Department of ENT
DMC&H, Ludhiana

Dr. Siddharth Bhargava
Associate Professor
Department of Paediatrics
DMC&H, Ludhiana

Date:

ENDORSEMENT BY THE HEAD OF THE INSTITUTE

This is to certify that the thesis entitled "**SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS"** is a bonafide research work done by **Dr. Deeksha Chawla** under the supervision of **Dr. Rohit Verma**, Professor, Department of ENT, **Dr. Manish Munjal,** Professor & Head, Department of ENT, **Dr. Siddharth Bhargava**, Associate Professor, Department of Paediatrics, in this institution.

Principal

Dr. Sandeep Puri
Principal
DMC&H, Ludhiana

Date:

COPYRIGHT

Declaration by the Candidate

I hereby declare that the Baba Farid University of Heath Sciences, Faridkot, shall have the rights to preserve & use this thesis in print or electronic format for academic/study purpose by the research scholar/students.

Signature

Dr. Deeksha Chawla
Department of ENT,
(Otorhinolaryngology)
Dayanand Medical College & Hospital,
Ludhiana, Punjab

Date:

© Baba Farid University of Health Sciences, Faridkot

Dedicated

to

my Family

&

Teachers

ACKNOWLEDGEMENT

This thesis not only represents my work at the keyboard, it is a milestone in my work of 3 years at this institute. My experience has been nothing short of amazing. I am highly grateful to the God Almighty for granting me strength to persevere and complete my thesis. I will always be grateful for all that I have.

I take this opportunity to thank all those who have helped in the successful completion of this work.

I am really indebted to my Supervisor and mentor, **Dr. Rohit Verma**, Professor, Department of Otorhinolaryngology, DMC & H, Ludhiana for being so supportive throughout this journey. His indelible help and fervent interest at all stages helped me a long way in completion of this project. His academic excellence, commitment to Otorhinolaryngology and readiness to teach is an example to all. I take great pride in expressing my gratitude to him.

With a deep sense of gratitude, I wish to convey my thanks to my esteemed teacher and co-supervisor, **Dr. Manish Munjal**, Professor and Head , Department of Otorhinolaryngology, DMC&H, Ludhiana who has been most helpful and supportive throughout my tenure. His able and mature guidance and ever willing help made the assigned task simpler. His dedication and hardwork has been a source of inspiration always. He has been a constant encourager.

I want to offer my sincere thanks to **Dr. Siddharth Bhargava**, Associate Professor, Department of Pediatrics, DMCH who has been a valuable support and a teacher in the true sense of the word. He has been a guiding light always.

I have been blessed with the calm to my chaos, My Husband **Dr Anshuman Dhawan**, without whom my journey would have been incomplete as well as tiresome. His companionship made me sail through this arduous journey smoothly.

Words are inadequate to express my sense of gratitude to my parents**, Mr.Naveen Chawla and Mrs.Kajal Chawla and Mr. Ajay Dhawan and Mrs. Shashi Dhawan** for their eternal guidance, immense love, unfailing attention and blessings that have made me whatever I am today.

I express my thanks to my lovely kinfolks **Dr Mehak S Munjal, Dr Suryaveer Singh, Ms. Ashima Sofat and Dr Rahul Chawla** for being my strength and being forever there in thick and thin.

My sincere thanks to my consultants, **Dr Sanjeev Puri And Dr Sahil Maingi** for their constant motivation and guidance and my colleagues**, Dr Shivam Talwar, Dr Salony Sharma, Dr Hardeep Kaur, Dr Lovleen Sandhu, Dr Manik Kaushal and Dr Rubaldeep Kaur** for their help and cooperation**.**

There will remain many whose names do not feature above. Their anonymity, however, in no way suggests that their contribution has gone unacknowledged. I tender my sincere apology and at the same time thank them for just being there when they were needed the most.

*My special thanks to our steno **Mrs Sreekala Pillai,** department of Otorhinolaryngology.*

*I am extremely thankful to our audiologists **Ms Parul** and **Mr Rajiv Ranjan***

*I am also indebted to **Ms. Namita** and **Mr Naveen Bhatia** for taking out time at any time of the day. This would not have completed without them.*

Dr. Deeksha Chawla

ABBREVIATIONS

AABR	AUTOMATED AUDITORY BRAINSTEM RESPONSE
AAOO	AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
AAP	AMERICAN ACADEMY OF PEDIATRICS
ABR	AUDITORY BRAINSTEM RESPONSE
AIIMS	ALL INDIA INSTITUE OF MEDICAL SCIENCES
ANSD	AUDITORY NEUROPATHY SPECTRUM DISORDER
AOAE	AUTOMATED OTOACOUSTIC EMISSION
ASHA	AMERICAN SPEECH LANGUAGE HEARING ASSOCIATION
ASSR	AUDITORY STEADY STATE RESPONSE
BERA	BRAINSTEM EVOKED RESPONSE AUDIOMETRY
CHL	CONDUCTIVE HEARING LOSS
CODEPH	COMMISSION FOR THE EARLY DETECTION OF NEONATAL HEARING LOSS
DPOAE	DISTORTED PRODUCT OTOACOUSTIC EMISSION
JCIH	JOINT COMMITTEE ON INFANT HEARING
LBW	LOW BIRTH WEIGHT
NHSP	NEWBORN HEARING SCREENING PROGRAMME
NICU	NEONATAL INTENSIVE CARE UNIT
NIH	NATIONAL INSTITUTE OF HEALTH
NIHCDC	NATIONAL INSTITUE OF HEALTH CONSENSUS DEVELOPMENT CONFERENCE
OAE	OTOACOUSTIC EMISSION
PCHI	PERMANENT CHILDHOOD HEARING IMPAIREMENT
SCBU	SPECIAL CARE BABY UNIT

SNHL	SENSORINEURAL HEARING LOSS
TEOAE	TRANSIENT EVOKED OTOACOUSTIC EMISSION
UNHS	UNIVERSAL NEONATAL HEARING SCREENING
VLBW	VERY LOW BIRTH WEIGHT
WHO	WORLD HEALTH ORGANISATION

ABSTRACT

Introduction: The development and maturation of hearing process begins in the fetal stage and progresses up to 6 months of age**.** Neonatal hearing screening is a contemporary method introduced for the evaluation of hearing in neonates at an early stage. As there is paucity of literature on the use of otoacoustic emissions (OAE) and Brainstem evoked response(BERA) tests for screening, hence we conducted a prospective observational study to screen the high risk neonates admitted in neonatal intensive care unit (NICU) using OAE and further BERA (wherever indicated).

Aims and Objectives: The primary objective of our study was to estimate the incidence of hearing loss in high risk neonates admitted in neonatal ICU in our hospital using otoacoustic emissions as a screening tool. Secondary objective was to determine the risk factors predictive of hearing impairment in these neonates.

Materials and Methods: This study was conducted in the neonatal intensive care unit (NICU) by the department of Otorhinolaryngology over a period of 1 year from 1st April 2021 to 31st March 2022. In this prospective observational study, 50 neonates with high risk factors admitted to NICU who conformed the inclusion criteria were enrolled. The neonates were subjected to a two staged OAE screening test and neonates who failed the OAE screening were tested with BERA.

Results: Out of 50 neonates enrolled in the study, 22 neonates (44%) passed the Stage I OAE. Remaining 28 neonates(56%) underwent Stage II OAE, out of which 17(34%) passed the Stage II screening. 11 neonates with overall OAE results as REFER were taken up for BERA. 3 neonates (6%) had hearing loss on BERA while in 8 neonates BERA was normal. The overall incidence of hearing loss in our study was 6%. The common risk factors in neonates with hearing loss were- low birth weight in all 3 neonates, perinatal asphyxia in 2 neonates, hyperbilirubinemia in 1 neonate, use of ototoxic medications in 1 neonate and requirement of mechanical ventilation >5 days in 1 neonate. The variables with a significant correlation with the results of OAE were APGAR score and perinatal asphyxia at a low APGAR score in our study.

Conclusion: The overall OAE results as PASS were seen in 39(78%) neonates and REFER in 11(22%) neonates. The results when confirmed with BERA showed hearing loss in 3(6%) neonates. Hence the incidence of hearing loss in our study was 6% APGAR score at 1 min and 5 mins and perinatal asphyxia at a low APGAR score at 1 min and at 5 mins had statistically significant correlation with the outcomes of OAE screening.

Keywords: Newborn hearing loss screening, Otoacoustic emissions, High risk neonates, Neonatal hearing loss

TABLE OF CONTENTS

S. NO.	TOPIC	PAGES
I	INTRODUCTION	1-8
II	AIMS AND OBJECTIVES	9
III	REVIEW OF LITERATURE	10-41
IV	MATERIALS AND METHODS	42-48
V	OBSERVATIONS AND RESULTS	49-110
VI	DISCUSSION	111-126
VII	SUMMARY AND CONCLUSIONS	127-128
	BIBLIOGRAPHY	129-138
	APPENDICES – I - PROFORMA - MASTER CHART	
	APPENDICES – II - APPROVAL LETTER OF THESIS PLAN - THESIS PLAN	

LIST OF TABLES

Table No	Title	Page No.
Demographic Profile		
1	AGE OF NEONATES	49
2	GENDER OF NEONATES	50
3	MATERNAL AGE (IN YEARS)	51
4	AGE OF FATHER (IN YEARS)	52
5	OCCUPATION OF MOTHERS	53
6	OCCUPATION OF FATHERS	54
7	AREA WISE DISTRIBUTION OF NEONATES	55
Clinical Profile		
8	BIRTH ORDER OF NEONATES	56
9	APGAR SCORE AT 1 MIN	57
10	APGAR SCORE AT 5 MINS	57
11	ANTENATAL HISTORY OF HYPERTENSION	60
12	ANTENATAL HISTORY OF PREECLAMPSIA	61
13	ANTENATAL HISTORY OF DIABETES	62
14	ANTENATAL HISTORY OF THYROID DISORDERS	63
15	ANTENATAL HISTORY OF EPILEPSY	64
16	ANTENATAL HISTORY OF SENSORINEURAL HEARING LOSS(SNHL)	65
17	MATERNAL HISTORY OF DRUG INTAKE	66
18	TYPE OF DELIVERY	67
19	PLACE OF DELIVERY	68
20	DISTRIBUTION OF FAMILY HISTORY OF SENSORINEURAL HEARING LOSS(SNHL) IN NEONATES	70
21	DISTRIBUTION OF HISTORY OF TORCH INFECTIONS IN NEONATES	71
22	DISTRIBUTION OF CRANIOFACIAL ANOAMLIES IN NEONATES	72
23	DISTRIBUTION OF LOW BIRTH WEIGHT IN NEONATES	73

24	DISTRIBUTION OF HYPERBILIRUBINEMIA IN NEONATES	74
25	DISTRIBUTION OF USE OF OTOTOXIC DRUGS IN NEONATES	75
26	DISTRIBUTION OF BACTERIAL MENINGITIS IN NEONATES	76
27	DISTRIBUTION OF PERINATAL ASPHYXIA AT A LOW APGAR AT 1 MIN IN NEONATES	77
28	DISTRIBUTION OF PERINATAL ASPHYXIA AT A LOW APGAR AT 5 MINS IN NEONATES	77
29	DISTRIBUTION OF NEED OF MECHANICAL VENTILATION ≥5 DAYS IN NEONATES	79
30	GENERAL PHYSICAL EXAMINATION	80
31	LOCAL EXAMINATION OF EAR	80
32	CSF GRAM STAINING/CULTURE SENSITIVITY RESULTS	81
33	BLOOD CUTURE RESULTS	81
34	INVESTIGATION RESULTS	82
35	OAE STAGE I RESULTS	84
36	OAE STAGE II RESULTS	85
37	OAE RESULTS (OVERALL)	86
38	BERA RESULTS	87
39	DISTRIBUTION OF HIGH RISK FACTORS IN NEONATES WITH HEARING LOSS	88
40	AGE OF NEONATES(RANGE, MEAN AND STANDARD DEVIATION)	89
41	STATISTICAL ANALYSIS OF AGE OF NEONATES.	89
42	STATISTICAL ANALYSIS OF GENDER	90
43	STATISTICAL ANALYSIS OF AGE OF PARENTS (RANGE, MEAN AND STANDARD DEVIATION)	90
44	STATISTICAL ANALYSIS OF AGE OF MOTHER WITH STUDY OUTCOMES.	91
45	STATISTICAL ANALYSIS OF FATHER'S OCCUPATION	92

	WITH STUDY OUTCOMES	
46	STATISTICAL ANALYSIS OF COMMUNITY DISTRIBUTION WITH THE OUTCOMES OF SCREENING	93
47	STATISTICAL ANALYSIS OF BIRTH ORDER	94
48	MEAN AND STANDARD DEVIATION OF APGAR SCORES.	95
49	A: STATISTICAL ANALYSIS OF APGAR SCORE AT 1MIN. B: STATISTICAL ANALYSIS OF APGAR SCORE AT 5 MINS	96
50	STATISTICAL ANALYSIS OF HISTORY OF HYPERTENSION WITH THE STUDY OUTCOMES	98
51	STATISTICAL ANALYSIS OF HISTORY OF PRE ECLAMPSIA WITH THE STUDY OUTCOMES	98
52	STATISTICAL ANALYSIS OF HISTORY OF DIABETES WITH THE STUDY OUTCOMES	98
53	STATISTICAL ANALYSIS OF HISTORY OF THYROID DISORDERS WITH THE STUDY OUTCOMES	99
54	STATISTICAL ANALYSIS OF HISTORY OF EPILEPSY WITH THE STUDY OUTCOMES	99
55	STATISTICAL ANALYSIS OF HISTORY OF SENSORINEURAL HEARING LOSS WITH THE STUDY OUTCOMES	99
56	STATISTICAL ANALYSIS OF PLACE OF DELIVERY	100
57	STATISTICAL ANALYSIS OF TYPE OF DELIVERY	100
58	STATISTICAL ANALYSIS OF FAMILY HISTORY OF SENSORINEURAL HEARING LOSS WITH STUDY OUTCOMES.	101
59	STATISTICAL ANALYSIS OF HISTORY OF TORCH INFECTIONS WITH STUDY OUTCOMES	102
60	STATISTICAL ANALYSIS OF HISTORY OF CRANIOFACIAL ANAMOLIES WITH STUDY OUTCOMES	103

61	STATISTICAL ANALYSIS HISTORY OF LOW BIRTH WEIGHT WITH STUDY OUTCOMES	104
62	STATISTICAL ANALYSIS OF HISTORY OF HYPERBILIRUBINEMIA WITH STUDY OUTCOMES	105
63	STATISTICAL ANALYSIS OF HISTORY OF USE OF OTOTOXIC MEDICATIONS WITH STUDY OUTCOMES	106
64	STATISTICAL ANALYSIS HISTORY OF BACTERIAL MENINGITIS WITH STUDY OUTCOMES	107
65	TABLE 65(I,II) : STATISTICAL ANALYSIS OF HISTORY OF PERINATAL ASPHYXIA AT A LOW APGAR SCORE WITH STUDY OUTCOMES I)PERINATAL ASPHYXIA AT LOW APGAR SCORE AT 1 MIN II) PERINATAL ASPHYXIA AT LOW APGAR SCORE AT 5 MINS	109
66	STATISTICAL ANALYSIS OF NEED FOR MECHANICAL VENTILATION ≥ 5 DAYS WITH STUDY OUTCOMES	110

LIST OF FIGURES

Figure. No	Title	Page No.
DEMOGRAPHIC PROFILE		
1	AGE OF NEONATES	49
2	GENDER DISTRIBUTION OF NEONATES	50
3	AGE OF MOTHERS (IN YEARS)	51
4	AGE OF FATHERS (IN YEARS)	52
5	OCCUPATION OF MOTHERS	53
6	OCCUPATION OF FATHERS	54
7	AREAWISE DISTRIBUTION OF NEONATES	55
CLINICAL PROFILE		
8	BIRTH ORDER OF NEONATES	56
9	APGAR SCORE AT 1 MIN AND 5 MINS	58
10	ANTENATAL PARAMETERS	59
11	ANTENATAL HISTORY OF HYPERTENSION	60
12	ANTENATAL HISTORY OF PREECLAMPSIA	61
13	ANTENATAL HISTORY OF DIABETES	62
14	ANTENATAL HISTORY OF THYROID DISORDERS	63
15	ANTENATAL HISTORY OF EPILEPSY	64
16	ANTENATAL HISTORY OF SNHL	65
17	MATERNAL HISTORY OF DRUG INTAKE	66
18	TYPE OF DELIVERY	67
19	PLACE OF DELIVERY	68
20	HIGH RISK FACTORS	69
21	FAMILY HISTORY OF SNHL IN NEONATES	70
22	DISTRIBUTION OF HISTORY OF TORCH INFECTIONS IN NEONATES	71
23	DISTRIBUTION OF HISTORY OF CRANIOFACIAL ANOMALIES IN NEONATES	72
24	DISTRIBUTION OF LOW BIRTH WEIGHT DISTRIBUTION IN NEONATES	73

25	DISTRIBUTION OF HYPERBILIRUBINEMIA IN NEONATES	74
26	DISTRIBUTION OF USE OF OTOTOXIC MEDICATIONS IN NEONATES	75
27	DISTRIBUTION OF BACTERIAL MENINGITIS IN NEONATES	76
28	DISTRIBUTION PERINATAL ASPHYXIA WITH LOW APGAR AT 1 AND 5 MINS IN NEONATES	78
29	DISTRIBUTION OF NEED FOR MECHANICAL VENTILATION ≥5 DAYS IN NEONATES	79
30	THE ILLUSTRATIONS DEPICTING THE FLOW OF EVENTS AND OUTCOMES DURING THE NEONATAL HEARING SCREENING	83
31	OAE STAGE I RESULTS	84
32	OAE STAGE II RESULTS	85
33	OAE TEST RESULTS (OVERALL)	86
34	BERA RESULTS	87
35	BIRTH ORDER IN OAE PASS	94

INTRODUCTION

Hearing and Language are one of the most significant aspects of human being.[1] The maturation and development of the structural part of the auditory system is known to begin in the first 20 weeks of gestation and that of the neurosensory part after 20 weeks. The auditory system is functional by the age of 25-26 weeks of intrauterine life. The period from 25 weeks of gestation up to 6 months of age is the most crucial phase for the neurosensory development of the auditory system.[2,3] Acquisition of hearing is essential for an individual's language, learning and cognitive development. Hearing loss at an early age of life can cause impairment in speech, language, learning, poor academic performance, maladjustments in social and personal behaviour and emotional difficulties.[4] Congenital hearing loss and subsequent language disability can be devastating for the child. WHO estimates that the people with hearing loss (defined as loss more than 40dB) worldwide, has risen from 120 million in 1995 to atleast 278 million in 2005 and to around 466 million in 2020 [1,5,6] In the year 2012, there were 360 million people in the world with disabling hearing loss (5.3% of the world's population) with 32 million (9%) of these being children. This increased to 34 million in 2020.[5] Such high rates of hearing loss increase the morbidity and disability of the paediatric age group at an early age. This alarms the need for screening of the newborns for any hearing loss. Newborn hearing screening programmes accepted worldwide are essential in the early identification, diagnosis and intervention in infants with hearing loss. If not detected at an early stage, it further hampers the speech, cognitive and language development.

It is a known fact that if identification of the hearing loss and its remediation begins at an early stage (< 6 months) and subsequent early corrective intervention is undertaken (<1 year), the cognitive skills and language can be developed.[7,8] As stated by the American Academy of Paediatrics, in a published 'position statement' in 1982, the screening of high risk infants should start by the age of 3 months and

rehabilitation to commence by 6 months of age.[9] A screening test hence becomes crucial for the timely detection of newborn hearing loss. The aim of a newborn hearing screening programme should be to detect severe to profound hearing loss and to reduce the age at which intervention is begun.[10] The National Institute of Health (NIH) gave a consensus statement in 1993 in which it is recommended that the universal newborn hearing screening should be conducted by the age of 3 months and also stated that otoacoustic emission might be the technology used for screening.[11] The screening test being used should be a feasible test which can be performed without a professional training. It should be cheaper, readily available and should not require a professional training and specialised laboratory setups. There are a plethora of subjective and objective testing modalities available for hearing evaluation. A subjective test is a test that requires the participation of the patient and his responses whereas an objective test detects the electrical responses and does not require a patient to participate. Subjective tests include pure tone audiometry, speech audiometry and reflex audiometry. These tests are not practical in newborns as subject's active participation is required. Objective tests include laboratory and bedside tests. For sensorineural hearing loss, tests like Otoacoustic Emission testing (OAE), Brain Stem Evoked Response (BERA), [also known as auditory brainstem response (ABR)], and Auditory Steady State Response (ASSR) can be used whereas for conductive hearing loss, Impedance audiometry is the objective test of choice.

OAE and BERA are the two feasible tests that can be conducted to assess sensorineural hearing loss but OAE is preferred over BERA as BERA involves shifting the baby to specialised laboratory facilities while OAE can be conducted bedside for the screening of babies.

Otoacoustic emissions (OAE) are the low intensity sounds produced by the outer hair cells of a normal cochlea and can be detected by a very sensitive microphone placed in the external ear canal and analysed by the computer.[12] The direction of the flow of the sound is reverse,

travelling from the outer hair cells → basilar membrane perilymph →oval window →ossicles →tympanic membrane →ear canal. The presence of otoacoustic emissions is indicative of a healthy ear and its absence that of the damaged ear and thus aiding in the assessment of cochlear function. Otoacoustic emission testing is also an efficient tool to differentiate between cochlear and retrocochlear pathologies.

There are two main types of OAE:

1. Spontaneous OAE which are present in healthy normal hearing people where hearing loss does not exceed 30dB.
2. Evoked OAE which is further of two types:
   A. Transient evoked OAE (TEOAE) which is evoked by clicks. In this a series of click stimuli are presented at 80-85dB.
   B. Distortion product OAE (DPOAE) In this two tones are simultaneously presented to the cochlea to produce distortion products. They have been used to test hearing in the range of 1000-8000 Hz.

OAE is one of the choices for screening for hearing loss.

- It involves the use of microphones, earphones or earplugs that are placed in the ear of the subject.
- Sensitive microphones and amplifiers are used to detect the emissions in response to the clicks or tone pairs at 80-86 dB sound pressure, typically responses to 260 stimuli captured over a time frame of first 20 ms after stimulus application in the 2f1-f2 paradigm.

There are a number of key definitions in relevance to screening tests for the quality assurance and audit of the screening programmes.[13] These include:

1. Coverage: Proportion of eligible population who complete the screen.
2. Screen positive: screening test that is greater than or equal to a specified cut off levels.

A) <u>True Positive</u>: who fail/refer the screen and have the disease

B) False Positive: Who fail/refer the screen but do not have the disease.

Certain factors interfering with the interpretations of otoacoustic emissions include ear canal debris, ambient sound, myogenic interference, infant's position, or activity, which may falsely influence the results of the screening tests.[14]

Such false positive results impose an emotional stress and negative impact over the psychological health of the parents.

3) Screen negative: screening test that is less than specified cut off levels.

A) True Negative: who pass the screen and donot have the disease

B) False Negative: Who pass the screen but have the disease.

According to the AIIMS Neonatology Protocols, a 'High Risk Neonate' is defined as the one with [15]: birth weight <1500gms, gestation <32 weeks, infants with birth weight of 1500 gms or more or gestation 32 weeks or more and

a. Intrauterine growth centile <3rd centile
b. Meningitis
c. Received mechanical ventilation for 48hrs or more
d. Hypoxic ischemic encephalopathy stage 2 or more
e. Major malformation
f. Inborn error of metabolism/chromosomal or genetic disorders/intrauterine infections
g. Symptomatic hypoglycemia
h. Symptomatic polycythemia
i. Retrovirus positive mother

Risk factors for neonatal hearing loss according to the Joint Committee on Infant Hearing Screening (JCIHS-2007), currently recommended by the Commission for the Early Detection of Neonatal Hearing Loss (CODEPH) are: [3]

1. Suspicion of hearing loss or developmental delay in the acquisition of speech, language.

2. A family history of permanent childhood hearing loss.
3. All children, with or without risk indicator, with a NICU admission greater than five days, including any of the following: extracorporeal oxygenation, assisted ventilation, ototoxic antibiotics (gentamicin, tobramycin), loop diuretics (furosemide). Also, regardless of the duration of admission: hyperbilirubinemia requiring exchange transfusion.
4. Intra-uterine infections such as cytomegalovirus, herpes, rubella, syphilis, and toxoplasmosis.
5. Craniofacial abnormalities, including those involving the auditory pavilion, auditory canal, and temporal bone malformations
6. Physical findings such as a strand of frontal white hair, associated with a syndrome that includes permanent sensorineural or transmissive deafness.
7. Syndromes associated with progressive or late-onset hearing loss or deafness, such as neurofibromatosis, osteopetrosis, and Usher syndrome. Other frequently identified syndromes include Waardenburg, Alport, Pendred, and Jervell and Lange-Nielsson.
8. Neurodegenerative diseases such as Hunter syndrome, sensorimotor neuropathies such as Friederich's ataxia and Charcot-Marie-Tooth syndrome.
9. Postnatal infections associated with sensorineural hearing loss including confirmed bacterial and viral meningitis (especially herpes and chickenpox viruses)
10. Head trauma, especially skull base trauma or temporary fracture requiring hospitalization.
11. Chemotherapy.

A brief summary of hearing loss and neonatal screening has been given in box 1,2 [16]

Box 1. Definitions of Hearing Loss

Hearing Loss: Any impairment in the ability to hear sounds at thresholds considered normal. For children, a pure tone threshold average of more than 15 dB at 500, 1000, 2000, and 4000 Hz is considered outside the normative range, with larger reductions in hearing levels classified by severity. Severity of hearing loss is categorized below.

Severity of Hearing Loss

Slight: hearing thresholds 16 to 25 dB

Mild: hearing thresholds 26 to 40 dB

Moderate: hearing thresholds 41 to 55 dB

Moderately severe: hearing thresholds 56 to 70 dB

Severe: hearing thresholds 71 to 90 dB

Profound: hearing thresholds more than 90 dB

Timing of Hearing Loss

Congenital: identified in the neonatal period.

Delayed-onset: identified after the neonatal period but attributed to etiologies present at birth.

Acquired: occurs after the neonatal period and is attributed to etiologies not present at birth.

Sensorineural hearing loss: due to injury or defect within the cochlea, cochlear nerve, or the brainstem pathways to the auditory cortex.

Conductive hearing loss: due to injury or defect within the external or middle ear, including the external auditory canal, tympanic membrane, middle ear cavity, and ossicles.

Mixed hearing loss: combination of sensorineural and conductive types of hearing loss.

Box 2. Newborn Hearing Screening

Technology Used to Perform Screening

Otoacoustic Emissions (OAEs)

Sounds produced by outer hair cells in the cochlea in response to acoustic signals in the ear. This non invasive test has different forms, known as transient evoked and distortion product OAE.

Auditory Brainstem Response (ABR)

A non invasive test of the integrity of the auditory pathway from middle ear, to cochlea, to the vestibulocochlear nerve, and to brainstem, where the response is measured; the ABR can be used as a pass/fail test for screening, or to identify the threshold (softest sound) at which sounds are heard.

Automated Auditory Brainstem Response (AABR)

The hearing screening version of ABR for infants in the neonatal intensive care unit (NICU).

Protocols

Normal Newborn Nursery

Hearing screening of full-term newborns usually involves the OAE. If the infant does not pass the screen in one or both ears (termed as refer for diagnostic testing), then they may undergo a second screening at their primary care provider visit or the birth hospital before the age of 1 month; a second screening refer should then cause the infant to undergo diagnostic ABR testing before the age of 3 months.

Neonatal Intensive Care Unit

Hearing screening of infants from the NICU usually involves the AABR; a refer usually results in a transitory evoked otoacoustic emission test to rule out auditory neuropathy spectrum disorder and a diagnostic ABR before the age of 3 months (corrected for gestational prematurity).

This study has primarily focussed on the high risk neonates admitted in the Neonatal ICU for the occurrence of hearing loss and its timely detection.

Early intervention to rehabilitate the neonates found to be having hearing loss was also advised to the parents. Otoacoustic emissions testing was used as the screening modality. With the screening facility available to the high risk neonate at commencement of development of skills, the rates of identification of the hearing loss, if any, can be hastened and the morbidities can be dismantled at an earlier stage. Such a study has not been conducted in our region and hence screening the neonates at hospital levels at an early phase of life would help to do a timely intervention, if required.

AIMS & OBJECTIVES

1. PRIMARY OBJECTIVE:

   To estimate the incidence of neonatal hearing loss in high risk neonates admitted in neonatal ICU in our hospital using otoacoustic emissions testing as a screening tool.

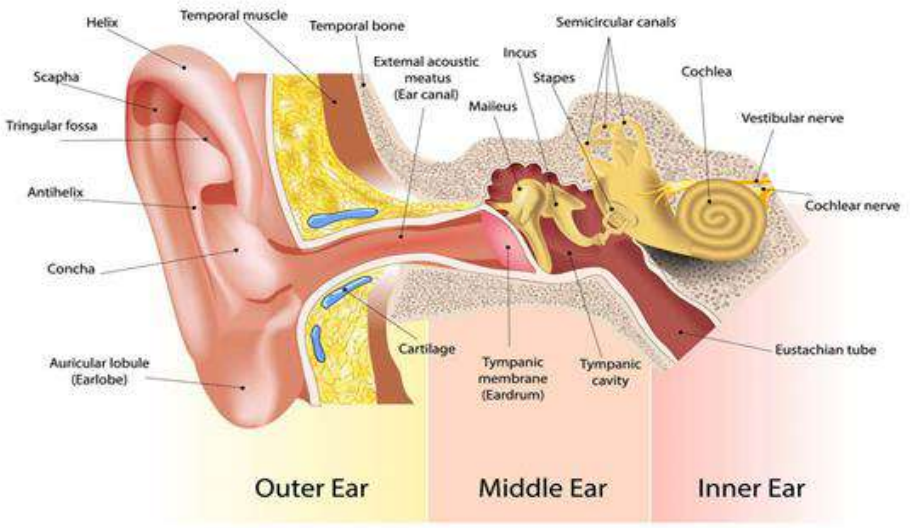
2. SECONDARY OBJECTIVE:

   To determine the risk factors predictive of hearing impairment in neonates.

REVIEW OF LITERATURE

I) **RELEVANT ANATOMY OF EAR:**[12,13]

The ear is divided into external, middle and inner ear. The external ear consists of pinna (also called auricle) and external auditory canal. The middle ear cleft comprises of middle ear cavity, eustachian tube and mastoid air cell system and the inner ear is composed of cochlea, vestibule and semicircular canals. (Shown in figure below)



Anatomy of Ear

II) **PHYSIOLOGY OF HEARING**:[12,13]

Hearing is an ability to perceive sound.

A) Mechanism of hearing :

The flow chart below shows the mechanism of conduction of sound from external to inner ear. (shown in figure below)

The auricle directs sound waves into the external auditory canal.

Sound waves strike the tympanic membrane causing it to vibrate back and forth. The distance it moves depends upon the intensity and frequency of the waves.

The central eardrum connects to the Malleus, which also starts to vibrate. This vibration is then transmitted to the Incus and Stapes

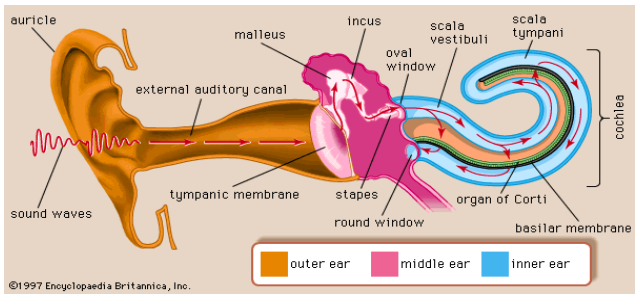
As the stapes moves back and forth, it pushes the membrane of the oval window in and out

The movement of the oval window sets up fluid pressure waves in the perilymph of the cochlea.

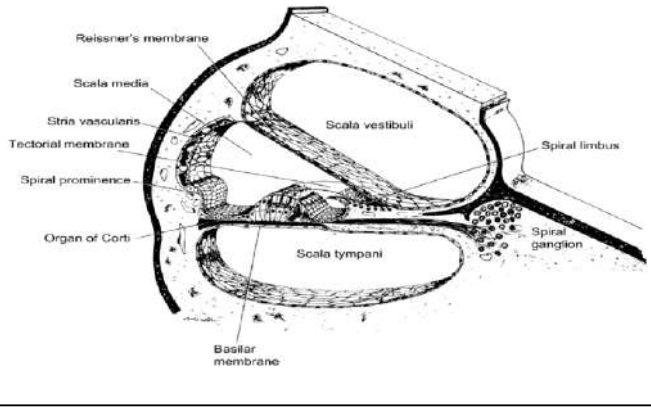
Pressure waves are transmitted to the round window, causing it to bulge outward

These waves in turn create pressure waves in the endolymph of the cochlear duct.

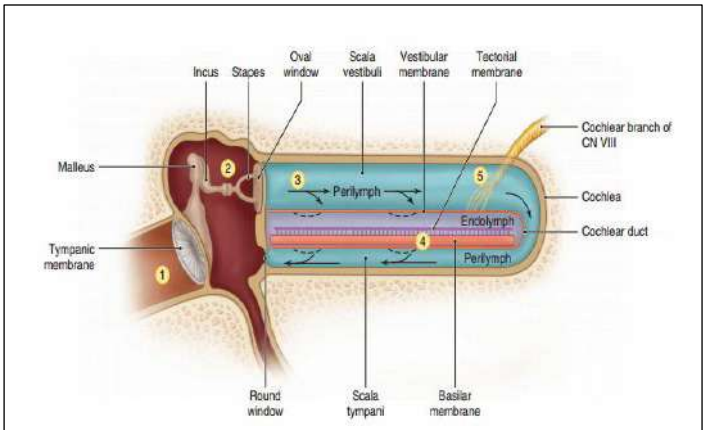
This causes the basilar membrane to vibrate, which moves the hair cells leading to receptor potentials and ultimately nerve impulses.



Mechanism of hearing



Human ear - Cochlea

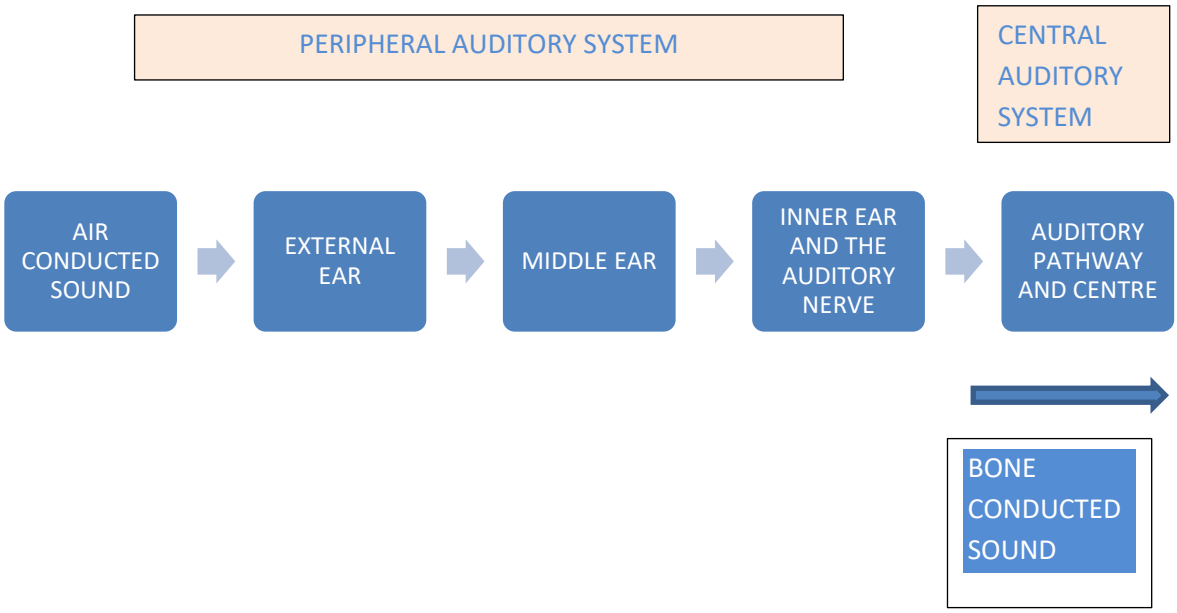


Fluids in inner ear

B) Auditory Pathway:

It comprises of the Central and Peripheral auditory system.

(As shown in figure below)



The Auditory System

CENTRAL PATHWAY:
(Shown in figure below)

Information from the Organ of Corti

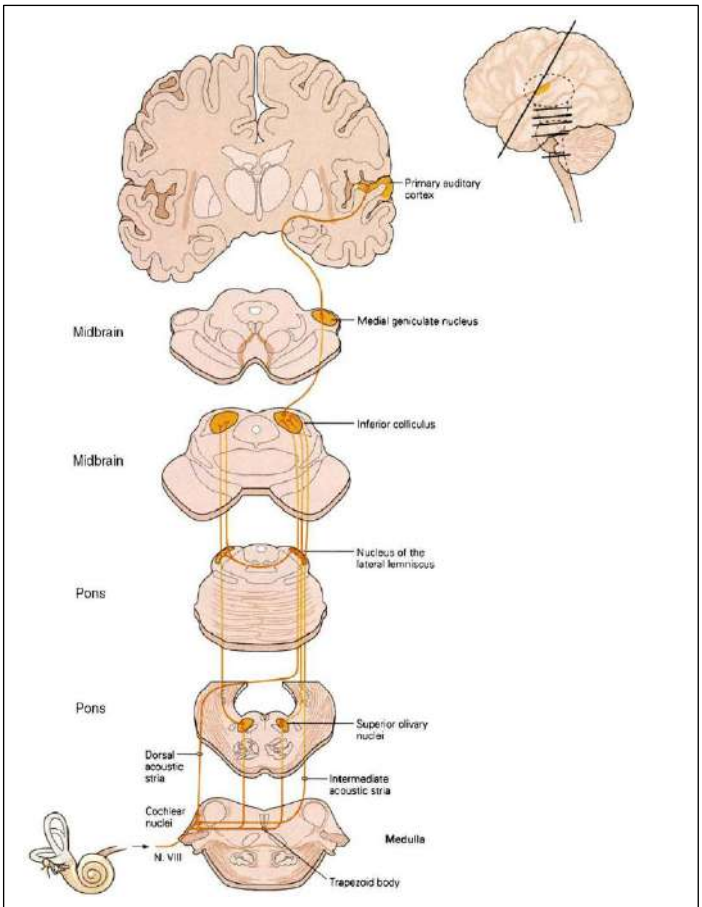
Spiral ganglions
and VIII CN afferents

Cochlear nucleus

Many crossing in the trapezoid body to the superior olive in the brain stem

Then all ascending fibers stop in the inferior colliculus in the midbrain and the medial geniculate body in the thalamus

The cortex in the superior temporal gyrus



Central Auditory Pathway

III) DEVELOPMENT OF HEARING: [17]

	Part of ear	Site of embryological development
1	External ear	1st and 2nd branchial arch and groove
2	Middle ear	1st and 2nd branchial arch and groove
3	Inner ear	Otocyst
4	Malleus and Incus	1st and 2nd branchial arch
5	Stapes	2ndbranchial arch and otic capsule

The external and middle ear are derived from the first and second branchial arches and grooves and grow throughout the puberty.

The inner ear which develops from the otocyst, reaches the adult size and shape by midfetal development. Malleus and incus are derived from the first and second branchial arches and stapes from the second branchial arch and otic capsule. The malleus and incus achieve the adult size and shape by 15th week of gestation and stapes by 18th week of gestation.

Pinna, ear canal and tympanic membrane continue to grow after the birth. The hearing in the fetus develops by 25 weeks of gestation.

IV) HIGH RISK NEONATES:

- According to the AIIMS Neonatology Protocols, a 'High Risk Neonate' is defined as the one with [15] :

1. Birth weight <1500gms
2. Gestation <32weeks
3. Infants with birth weight of ≥1500 gms or gestation ≥ 32 weeks and
    a. Intrauterine growth centile <3rd centile

b. Meningitis
c. Received mechanical ventilation for 48hrs or more
d. Hypoxic ischemic encephalopathy stage 2 or more
e. Major malformation
f. Inborn error of metabolism/chromosomal or genetic disorders/intrauterine infections
g. Symptomatic hypoglycemia
h. Symptomatic polycythemia
i. Retrovirus positive mother

- The Joint Committee on Infant Hearing (JCIH) is a collaboration of representatives from audiology, otolaryngology, pediatrics, and nursing. It was built in the late 1969. The American Speech Language Hearing Association (ASHA), the American Academy of Ophthalmology and Otolaryngology (AAOO) and the American Academy of Pediatrics (AAP) form the essential bodies of JCIH.[18] The position statement issued in 1972 by the Joint Committee, delineated the high risk factors for hearing loss.

The JCIH states the following high risk factors:

1. Family history of hereditary childhood sensorineural hearing loss.
2. In utero infections (Toxoplasmosis, Rubella, Cytomegalovirus, Herpes simplex virus infections and Syphilis)
3. Craniofacial anomalies.
4. Birth weight <1500 g.
5. Hyperbilirubinemia at serum levels requiring exchange transfusion.
6. Ototoxic medications.
7. Bacterial meningitis.
8. Perinatal asphyxia (Apgar of 5 at 1 minute or of 6 at 5 minutes).
9. Mechanical ventilation lasting 5 days or longer.
10. Stigmata or other findings associated with a syndrome known to include a sensorineural hearing loss.

V) HEARING LOSS IN CHILDREN: [13]

Permanent childhood hearing impairment (PCHI) is defined as a confirmed permanent bilateral hearing impairment of ≥40 dBHL averaged over the frequencies of 0.5,1,2,4kHz in the better hearing ear. A PCHI can be conductive, sensorineural or mixed. It can either be Prelingual Deafness (before the child attains the ability to speak) or Postlingual Deafness (after the child attains ability to speak).

Classification of degree of hearing loss:

Degree of hearing loss has been broadly classified by various systems into categories as described below.

Classification of degree of hearing loss:

dB HL	WHO	NHSP	BSA[13]	ASHA[12]
Normal	≤25	≤20	<20	–10 to 15
Slight	26 to 40			16 to 25
Mild		21 to 39	20 to 40	26 to 40
Moderate	41 to 60	40 to 69	41 to 70	41 to 55
Moderately severe				56 to 70
Severe	61 to 80	70 to 94	71 to 95	71 to 90
Profound	≥81	≥95	>95	>90

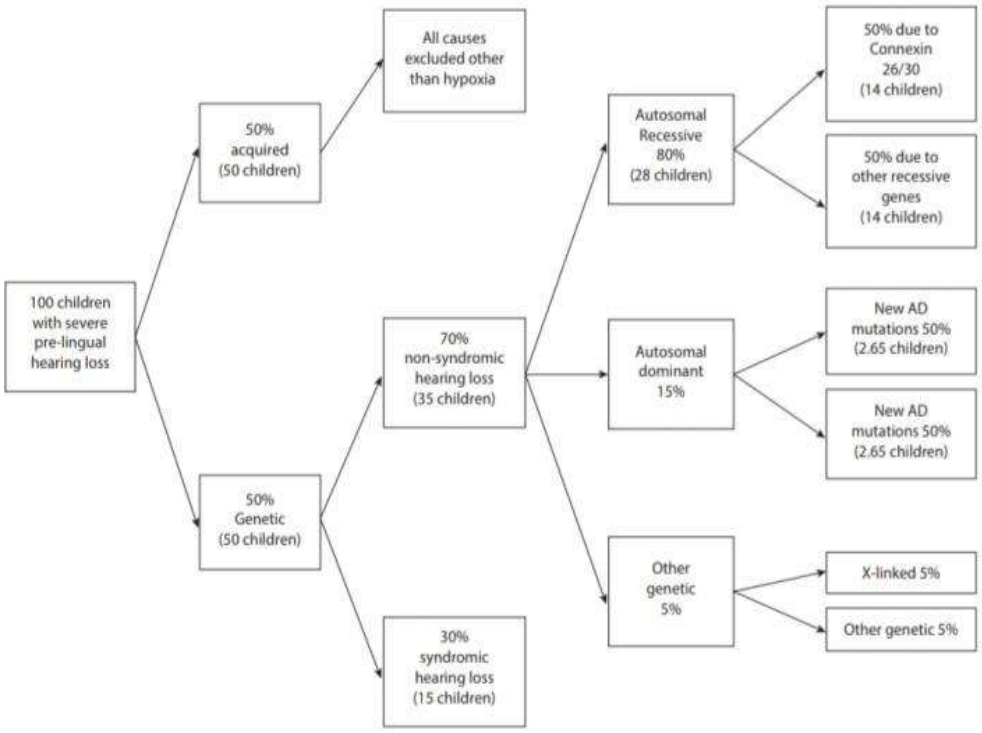
WHO World Health Organisation; NHSP Newborn Hearing Screening Programme; BSA British Society Audiology; ASHA American Speech-Language Hearing Association.

The etiology of hearing loss can be congenital or acquired. Causes of congenital hearing loss are broadly divided into genetic and non genetic causes.[19] Further more the genetic causes are divided into syndromic (30%) and non syndromic (70%). The non syndromic etiology can either be autosomal dominant (20%) or autosomal recessive (80%). The mutation in the connexin encoding gene is thought to be responsible for half of these nonsyndromic causes.[20] The most common non genetic cause of hearing loss is cytomegalovirus infection, 0.2-2.5% of all new borns globally. The most common type of hearing loss caused by CMV infections is the

sensorineural hearing loss (usually severe to profound type).[21]Other infections associated with hearing loss include the TORCH infections namely toxoplasmosis, rubella, syphilis and herpes.[22]

Leal et al in one of their studies also proved zika virus to be one of the etiological factor for hearing loss in children.[23,24]

Causes of aquired hearing loss are Perinatal, Postnatal and Idiopathic (as tabulated below)


100 children with severe pre-lingual hearing loss
50% acquired (50 children)
50% Genetic (50 children)
All causes excluded other than hypoxia
70% non-syndromic hearing loss (35 children)
30% syndromic hearing loss (15 children)
Autosomal Recessive 80% (28 children)
Autosomal dominant 15%
Other genetic 5%
50% due to Connexin 26/30 (14 children)
50% due to other recessive genes (14 children)
New AD mutations 50% (2.65 children)
New AD mutations 50% (2.65 children)
X-linked 5%
Other genetic 5%

Causes of hearing loss

Causes of hearing impairment		
Congenital disorders		
Genetic	Syndromic	Autosomal recessive
		Autosomal dominant
		X-linked
		Mitochondrial
	Non-syndromic	Autosomal recessive
		Autosomal dominant
		X-linked
		Mitochondrial
Non-genetic	Congenital rubella syndrome	
	Cytomegalovirus First-trimester maternal infections (viral or bacterial)	
	Congenital syphilis Toxoplasmosis Hyperbilirubinemia Low birth weight Birth asphyxia Drugs during pregnancy, such as aminoglycosides, some antibiotics, anti-epilleptic drugs, cytotoxic drugs, antimalarial drugs, and diuretics. Maternal substance abuse e.g. alcohol, recreational drugs Maternal endocrine disorders e.g. diabetes mellitus, thyroid dysfunction 'Third window' disorders (see text, aetiology often unknown, may be congenital or acquired)	
Acquired causes		
Perinatal	Hypoxia	
	Hyperbilirubinemia	
Postnatal	Infections	Chronic otitis media
		Herpes Meningitis
		Mumps
		Measles Toxoplasmosis Varicella
		HIV
	Ototoxic drugs	
	Trauma / noise exposure	
	Neoplastic disease Foreign bodies in ear canal Immune system disorders	
Idiopathic		

Causes of hearing loss

According to Scott et al, there are three major risk factors for hearing loss [13]:

- History of treatment in neonatal intensive care unit (NICU) or special care baby unit (SCBU) for more than 48hrs.
- Family history of early childhood deafness.
- Craniofacial anomaly associated with hearing loss (cleft palate for eg.)

One or more of the risk factors associated with about 60% of congenital bilateral hearing loss of moderate or higher degree include 29.3% NICU, 26.7% family history and 3.9% craniofacial anomaly. In the Wessex study, 8.1% newborns with fulfilled high risk criteria amongst the 25,000 newborns screened.[25] Davis and Wood showed that babies admitted to NICU for more than 48hrs were 10.2 times

more likely to have a permanent hearing loss than a patient not admitted in NICU.[26]

Fortnum and Davis showed that the prevalence increased 14 folds for children with family history of hearing loss.[27,28]

Other perinatal causes of hearing loss stated by JCIH as discussed earlier are inclusive of prematurity, use of ototoxic drugs, hyperbilirubenimia, mechanical ventilation, asphyxia, low APGAR scores and prematurity.[18]

Ototoxic drugs like aminoglycosides (widely used in a NICU setting as a first line antibiotic) are known to cause cochlear and vestibular damage [29].

- Gentamycin and tobramycin are the most vestibulotoxic agents.
- Neomycin, kanamycin and amikacin are cochleotoxic agents.

The hair cells are known to get damaged by the action of the ototoxic drugs, affecting the higher frequencies first followed by the lower frequencies.[30,31]

Hyperbilirubinemia is another potential risk factor that is known to cause hearing loss and further damage to the brainstem auditory nuclei selectively. It may also pose damage to the auditory nerve and ganglion cells. [32]

Auditory Neuropathy Spectrum Disorder (ANSD) is one such entity that is known to occur in newborns with high bilirubin levels [33,34] As recommended by the American Academy of Pediatrics, the average noise levels in neonatal units should be below 45 dB and transient sounds should not exceed 65 dB.[35-38] Adequate oxygenation and perfusion is a necessity for the normal perfusion and functioning of the cochlear function. Lower oxygen levels and hypoxia can cause irreversible cellular damage to the cochlear function by affecting the outer hair cells and stria vascularis.[39]

Robertson et al. showed that mechanical ventilation and prolonged oxygen supplementation were associated with high prevalence of permanent hearing loss in extremely premature infants [40].

Hille et al similarly described that assisted ventilation for more than 5 days is an independent risk factor for hearing loss.[41]

VI) ASSESSMENT OF HEARING LOSS IN CHILDREN:[13]

- Behavioural Observation Audiometry
- Visual Reinforcement Audiometry
- The Distraction Test
- Performance Testing (Play Audiometry)
- Pure Tone Audiometry
- The Cooperative Test
- Speech Discrimination Test
- Electrophysiological Test

1. <u>Behavioural Observation Audiometry</u>

The changes in the activity are observed in response to the sound stimulus.

The developmental age: 0-6 months

Response behaviours include eye widening, eye blinking (auropalpebral reflex), alteration in sucking response, arousal from the sleep, startle or shudder of the body or definite movement of arms, legs or body. By 4-7 months, lateral inclination of head towards the sound can be noted.

Method: a sound stimulus is given for <2 seconds, 15 cm from the child's ear, in a horizontal plane and away from the peripheral vision.

The reliability of this test as a diagnostic test is less.

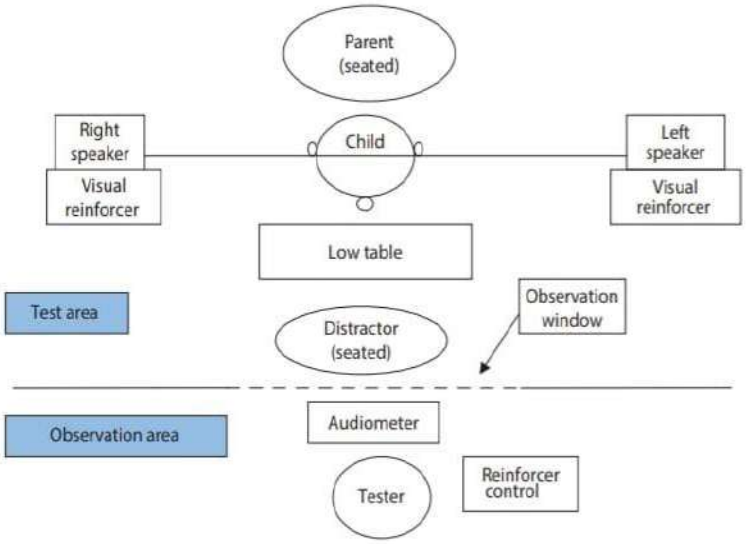
2. <u>Visual Reinforcement Audiometry</u>

Key developmental age: 5-36 months

This term was first introduced by Liden and Kankkunen [42]

This test is primarily done for hearing assessment in preschool children and for infants once they have reached the developmental stage of being able to sit unsupported or with minimal support, i.e. at 5–7 months. The test room should be sound-treated to ensure low levels

of ambient noise (ISO 8253).This should be verified using a sound level meter. The room should be adequately sized (recommended minimum of 6m×4m), ideally with the ability to dim the lighting to permit enhancement of the illuminated vision reward. For sound field testing, the loudspeakers should be placed at 90° from the child and at the same height as the child's head at a distance of at least 1m from the ear. The first step is to establish the conditioned response. Appropriately calibrated frequency modulated (warble) tones, ling sounds or narrow-band noise (NBN) stimuli may be employed via sound field, insert earphone, head-phone or bone conductor, according to the clinical priority. Once the conditioning of the response is done, the aim is to establish the minimal response level (MRL)
for each frequency. A typical order of stimulus presentation could be: 2kHz – 500Hz – 4kHz – 1kHz or 1kHz – 4kHz – 500Hz – 2kHz.
The figure below shows the test arrangement.

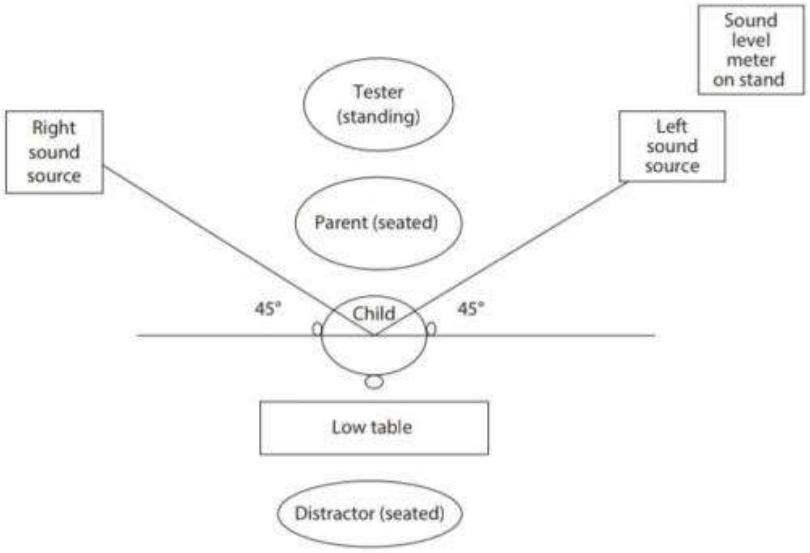


Arrangement of the Visual Reinforcement Audiometry[13]

3. The Distraction Test

The infant distraction test was first described by Ewing and Ewing[43] in 1944.The test is based on the principle that the normal response observed when sound is presented to an infant is a head turn to locate the source of sound. The test is suitable at 6–18 months, corresponding to the stage when the child can sit erect

unsupported and perform head turns in a horizontal plane. The sound stimulus should be presented in the horizontal plane to the ears at an angle, set back 45° between 1m and 15cm from the child's ear. A wide range of sound stimuli can be used to elicit a response, including voice (e.g. unforced 'S' = high frequency, or hum = low frequency), musical toys, calibrated high-frequency rattle, NBN or warble tones. The normal response expected is a full head turn in the direction of the sound. The figure shown below shows the test arrangement.



Arrangement of the Distraction Test[13]

4. Performance Testing (Play Audiometry)

Key developmental age: 2–5 years

The performance test was first described by Ewing and Ewing[43] as a transitional technique suitable for children from 2.5 years and in some cases younger, until cooperation with pure tone audiometry can be achieved. It follows the simple principle that the child is conditioned to wait for a sound and then to respond with a play activity. Toys which involve a simple repetitive activity are most suitable, such as the classic 'men in a boat', balls on sticks, or pegs in a board. The conditioning sequence starts with the tester engaging the child's attention by holding the response item (e.g. the wooden man) poised

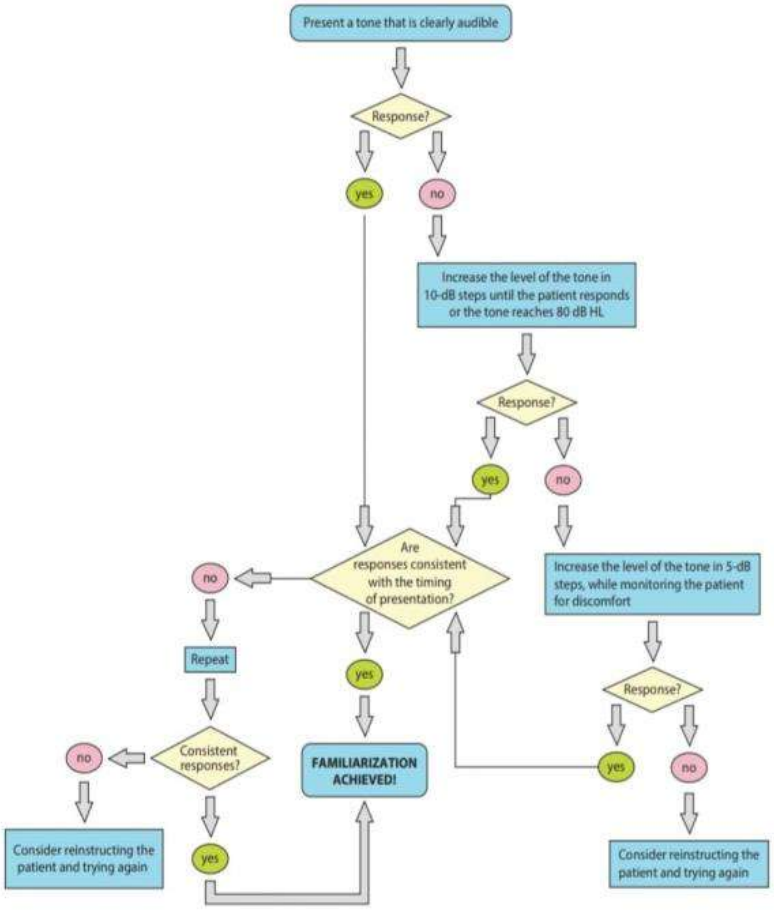
waiting in front of the child. After a few seconds, a suprathreshold sound stimulus is presented and the tester responds by an appropriate activity, such as placing the man in the boat. This sequence is repeated several times to create a conditioned reflex in child. Once it has been established, using the 10 dB down, 5 dB up technique the MRL is determined.

5. Pure Tone Audiometry

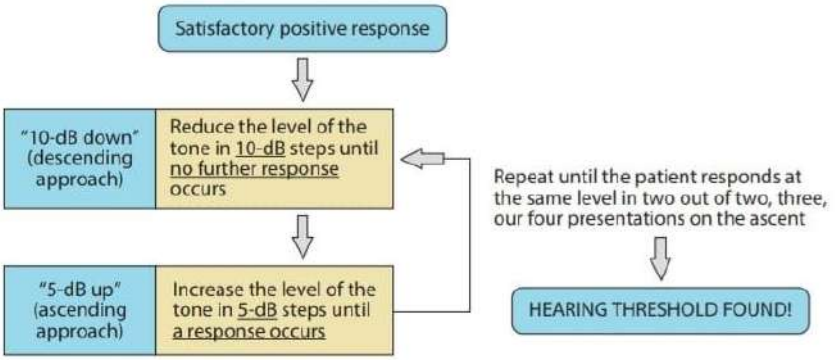
Key developmental age: beyond 3 years.

Limitation: Not a useful test for neonatal screening as it is a subjective test and results depend upon the response of the subject to the stimuli provided.

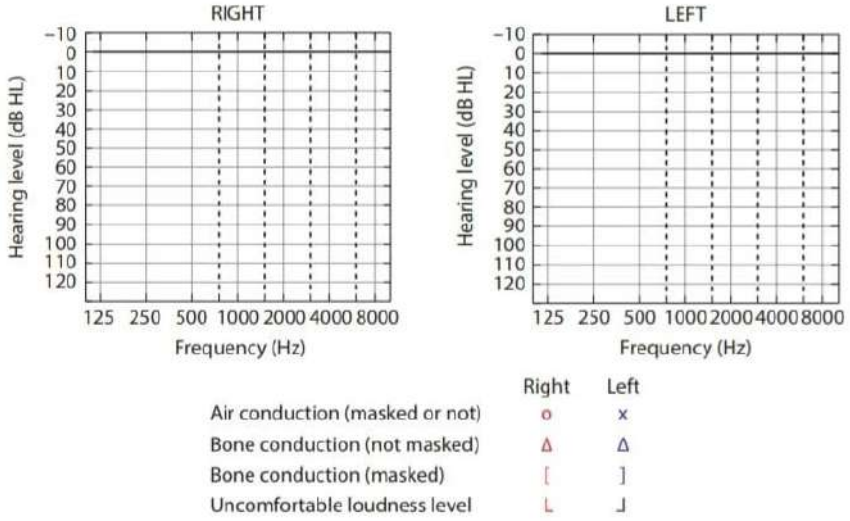
The principal equipment for deriving the clinical audiogram is the audiometer. When performing PTA, the tester fits the transducers to the patient, and the patient is instructed not to move or hold the transducers. The examiner should check for any discomfort caused by the transducers. The use of air conduction (AC) and bone conduction (BC) transducers seeks to separate the conductive (outer and middle ear) and sensory (cochlear) components of hearing loss. Signals presented from headphones or insert earphones are transmitted by AC. In BC testing, a bone vibrator is positioned on the mastoid behind the pinna or sometimes on the forehead, with a known force so that mechanical vibrations are coupled to the skull bone to stimulate the cochlea through bone transmission. It is started in the better ear and the threshold for each of the pure tones is measured before moving to the next pure-tone test frequency. Traditionally, the order of the pure tones presented to the patient is: 1000Hz, 2000Hz, 4000Hz, 8000Hz, 500Hz and 250Hz.' It is known as Bracketing technique or the '10 dB down, 5 dB-up technique'.



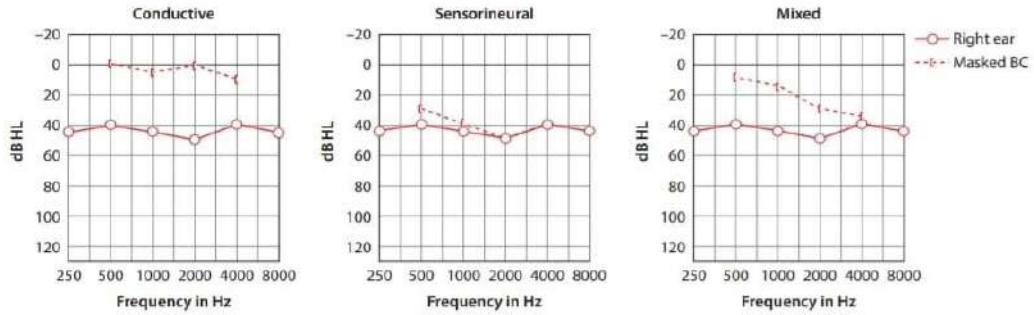
Procedure to achieve familiarisation with the task [13]


Satisfactory positive response
"10-dB down" (descending approach)
Reduce the level of the tone in 10-dB steps until no further response occurs
"5-dB up" (ascending approach)
Increase the level of the tone in 5-dB steps until a response occurs
Repeat until the patient responds at the same level in two out of two, three, our four presentations on the ascent
HEARING THRESHOLD FOUND!

Method to measure threshold



Audiogram and its symbols used



Types of hearing losses on an audiogram

6. The Cooperative Test

Linguistic developmental level: 18–30 months.

The cooperative test is another test originally described by Ewing and Ewing[43]. It requires the child to discriminate three different simple instructions, for instance having been handed a small toy, asked to 'give it to Mummy' or 'give it to teddy' or 'give it to baby'. Starting at a suprathreshold level, the voice is then dropped and visual clues removed by covering the mouth. A child with normal hearing may discriminate the instruction at 35–40dB.

7. Speech Discrimination Test

Linguistic developmental level: 30 months onwards

A wide range of speech discrimination assessments are available and may be valuable in verifying hearing thresholds and in assessing the effectiveness of hearing aids or auditory implants. The choice of test will depend on the child's age, stage of linguistic development and home language and may be influenced by non-auditory factors such as access to language, specific language impairment and cognitive level.

8. Electrophysiological Tests

- Otoacoustic Emissions (OAE)
- Brainstem Evoked Response Audiometry(BERA)

➢ Otoacoustic Emissions (OAE)

OAE are predominantly low intensity sounds produced by the outer hair cells of a normal cochlea and are assessed by a sensitive microphone or earphone placed in the ear canal and is analysed in a computer. OAE are produced by the healthy outer hair cells and absent in case of cochlear dysfunction. The direction of the flow of the sound is reverse, traveling from the outer hair cells → basilar membrane →perilymph →oval window →ossicles →tympanic membrane →ear canal. It is easy to perform, doesn't need an expertise, cost effective and less time consuming. It is an objective test. It takes about 1-5 minutes and can be performed in a sleeping or awake neonate. The interpretation of the test is in the form of PASS or REFER. PASS is suggestive of a normal ear and REFER may either indicate an abnormal ear or a false positive test.

Types of OAE:

1. Spontaneous OAE which are present in healthy normal hearing people where hearing loss does not exceed 30dB.

2. Evoked OAE which is further of two types:

A) Transient evoked OAE (TEOAE): It is evoked by clicks. In this a series of click stimuli are presented at 80-85dB.

B) Distortion product OAE (DPOAE): In this two tones are simultaneously presented to the cochlea to produce distortion products.

- Brainstem Evoked Response Audiometry(BERA)

It is a non invasive test which involves the placement of electrodes over the various anatomical sites. A non-inverting electrode is placed over the vertex of the head and inverting electrode placed over the ear lobe or mastoid prominence. One more ear thing electrode is placed over the forehead. The principle of this test is to elicit brainstem responses to auditory stimulation by clicks and tone bursts. The integrity of central auditory pathways with the VIII CN, Pons and Midbrain is assessed. In this method the electrical potentials are generated in response to the several click stimuli or tone bursts and picked up from the vertex by surface electrodes. A hearing sensitivity in the range of 1000-4000Hz is measured. A normal person generates seven waves in first 10ms. The first three waves are most stable and used for the measurement. The waves are studied for absolute latency, interwave latency (between I and V) and their amplitude.

VII) NEONATAL HEARING SCREENING:

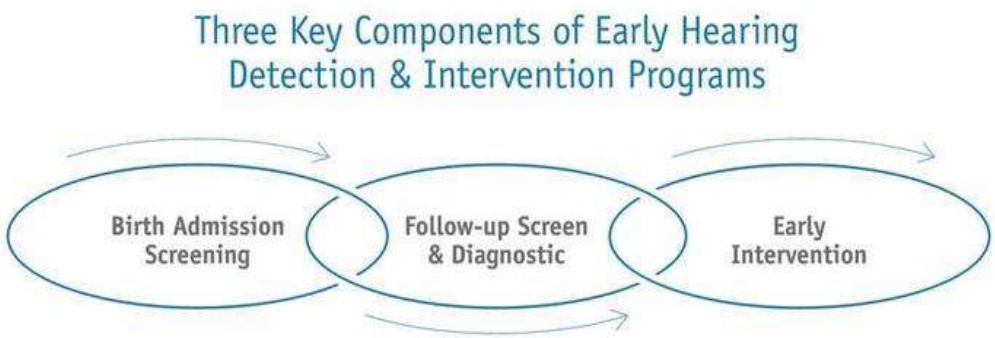
- **Historical significance**

With the introduction of the multiple programmes and schemes over the past three decades, there has been an immense progression in the opportunities to identify the neonatal hearing loss and provide these children with the age appropriate linguistic and communication skills.[44] Universal Newborn Hearing Screening (UNHS) is one such initiative to timely screen the neonates and do the needful intervention at an early phase of life. In the era of no definitive neonatal screening modality, neonates remained undiagnosed and the hearing loss could only be identified when any other developmental delay in milestones appeared. As we go back to the historical era, the fascination leads us to the first population-based programs in the United States in the mid-1990s.More than 50 years before the introduction of modern UNHS programs, great emphasis was being made in the fields of otolaryngology and

education of deaf. One such initiative was the use of objective electro-physiological assessments by Sir Alexander and Lady Ethel Ewing for the empowerment of newborn screening. The argument was to create'… an urgent need to study further and more critically methods of testing hearing in young children', with the aim of identifying deafness within the first year of a child's life; noting that education needed to commence '…at the earliest age that the diagnosis of deafness can be established'.[45] From that time, the desideratum of newborn screening became a common feature for detection of hearing loss. In 1956, Erik Wedenberg gave one of his finest works by the use of tuning forks, percussion sounds, pitch pipes, and cowbells to screen the neonates for hearing loss. The concept was based upon the auropalpebral or the cochleopalpebral reflex (An involuntary blink of the eye caused by contraction of the orbicularis oculi muscle in response to loud sounds). Newborns were provoked with the stimuli and reflexes recorded. In their opinion it is possible to provoke the infants before the age of 6 months as beyond that significant age it becomes difficult to generate these reflexes in response to stimuli.[46] By the 1960's great audiologists and practitioners like Marion Down and Steritt developed the behavioural observation screening programmes (Behavioural observational audiometry BOA) and focussed primarily on the screening of newborns at an early age.[47,48] They used a narrow band of noise centred on 3 kHz at an intensity of 90 dB and appointed a group of volunteers for the observation of 'blinking or startle' responses with respect to the stimuli. Between 1965 and 1967 around 17,000 babies in Denver hospitals were screened with an aim to identify the children with hearing thresholds greater than 65 dB. 17 infants were found to have hearing loss. This was one of the first practical implication of UNHS Model.[47,48]

The formulation of JCIH with the objectives of the early identification of children with or at-risk for hearing loss and newborn hearing screening, led to the revolutionary change in the series of neonatal hearing.[18] The American academy of paediatrics in 1982, also stated in a position

statement that the screening of high risk infants should start by the age of 3 months and rehabilitation to commence by 6 months of age. In 1994, the JCIH endorsed the goal of universal detection of hearing loss in newborns and infants. It stated that all infants with hearing loss should be identified before 3 months of age and receive intervention by 6 months.[9,18]


Three Key Components of Early Hearing Detection & Intervention Programs
Birth Admission Screening
Follow-up Screen & Diagnostic
Early Intervention

Components of neonatal hearing screening by JCIH [18]

The enterprise of National Institute of Health passed as a Consensus statement in 1993 stated the concept of "1-3-6" as the monthly milestones for screening, diagnosis and intervention and also stated that otoacoustic emission (OAE) might be the technology used for screening.[11,49] The National Institutes of Health Consensus Development Conference (NIHCDC) on Early Identification of Hearing Impairment was summoned in 1993 with the objectives of :(1) The merits of timely identification and demerits of late identifications of hearing impairments.(2) Whom and when to screen. (3) The advantages and disadvantages of the screening techniques available then.(4) Preferred model for screening and when to call for follow up.(5) Diagnosis and Management of hearing impairment in infants and young children. The Conclusion of the NIHCDC panel stated [11]: (1) All infants admitted to the neonatal intensive care unit must be screened for hearing loss prior to discharge.(2) Universal screening to be implemented for all infants within the first 3 months of life.(3) The preferred model for screening should begin with an evoked otoacoustic emissions test and should be followed by an auditory brainstem

response test for all infants who have failed the evoked otoacoustic emissions test.(4) Comprehensive intervention and management programs must be an integral part of a universal screening program. (5) Universal neonatal screening should not be a replacement for ongoing surveillance throughout infancy and early childhood.(6) Education of primary caregivers and primary health care providers on early signs of hearing impairment is essential.

- **Need for neonatal screening:**

The incidence of hearing loss in neonates remained unknown prior to the advent of the concept of neonatal screening. White KR et al in their study stated that the permanent hearing loss of greater than 25 decibel hearing level in poorer ear is present in at least 4 (1.1 to 6) per 1000 infants.[50] Abdullah A et al and Asma A et al conducted independent studies and concluded the prevalence of hearing loss in high risk infants to be between 2.5 and 10 %.[51,52]

Nagapoornima P et al conducted a study and showed the incidence of hearing impairment in neonates (at risk and not at risk) in India between 6-60 per 1000 neonates with an average of 4 per 1000.[53] Another survey by Ansari MS et al showed 4 out of 1000 infants born, had hearing loss.[54] According to the several studies conducted it has been concluded to screen the neonates at an early stage of life. Screening must begin by the age of 3 months and intervention by 6 months of age.[9]

One of the most published facts about the children with hearing loss is that of their lower academic performance in comparison to the children with no hearing loss. This statement by Meadow was talked about by her in one of her texts.[55,56] She quoted, 'It was looked that the gap particularly with respect to the lexical-semantic and morpho-syntactic abilities in spoken language, and particularly for children with more severe and profound hearing loss.'

Another longitudinal study conducted by White and White on developmental receptive and language skills of 46 children with hearing loss. The children were observed until they entered the preschool

program. By the age of 3 years, only 50 % of children could attain the skills in comparison to a normally developing child at 1 year of age.[57]

Paul et al were the active members in the meeting on formulation of national consensus guidelines on developmental disorders.[58] It was organized by the Indian Academy of Pediatrics in Mumbai, on 18th and 19th December, 2015. The primary agenda of the meet was to provide guidelines on newborn hearing screening in India.
The key messages stated by them were:

- Hearing loss should be screened preferably before 1 month of age.
- Universal neonatal screening rather than targeted 'high risk' screening is ideal.
- If abnormal OAE detected, it is repeated at 6 weeks or on the first immunization visit. If again abnormal, ABR is done for confirmation followed by full audiological evaluation and remediation with hearing aids.
- All NICU babies should undergo ABR testing to rule out auditory dys-synchrony/ auditory neuropathy.

This primarily focussed on the early screening of neonates before 6 months of age and preferably below 3 months of age.

- **<u>Protocol for Neonatal Screening:</u>**

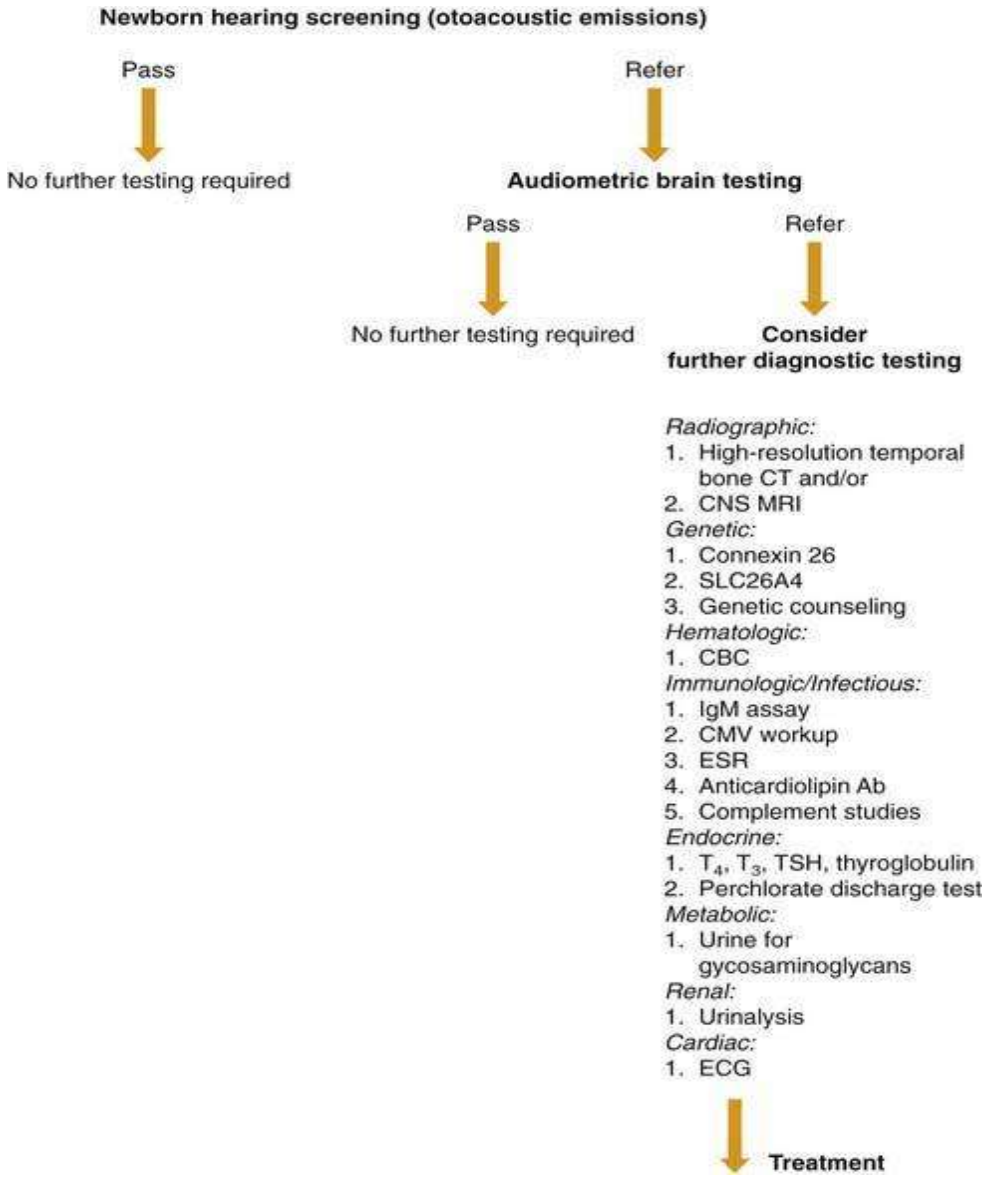
The National Hearing Screening Programme (NHSP) screening declared a standard protocol for the screening of well babies and the babies who have spent 48hours or more in NICU/SCBU.[59,60] It also specified a standard set of techniques and protocols that can be used.

- Well babies: "A two-stage two-technology protocol" is used. STAGE 1: Transient AOAE screen, with a maximum of two attempts

- STAGE 2: An AABR screen, if required. INTERPRETATION: A pass in both ears using AOAE or a pass in both ears on AABR, constitutes an overall screen pass.
- Babies cared for in NICU or SCBU for more than 48 hours: undergo a transient AOAE screen and an AABR screen.

INTERPRETATION: Those that fail the AABR screen in one or both ears are considered to be screen referrals and are referred for audiological assessment. Babies that pass the AABR screen in both ears and fail the AOAE screen in both ears are included in the risk-factor group and receive a follow-up at 8 months.

ALGORITHM FOR NEWBORN SCREENING as quoted in the Nelson book of paediatrics[17] has been shown in the flowchart below.



NEONATAL SCREENING ALORITHM

VIII) ROLE OF OTOACOUSTIC EMISSIONS TESTING IN NEONATAL SCREENING:

Otoacoustic emissions are the low intensity sounds generated from the outer hair cells of the inner ear. The story of acoustics begins with the Guiseppe Tartini, a violinist, who was the owner of first violin and appointed as the Maestro di Capella at the Basilica di Sant 'Antonio in Padua by 1721.[61-63] With his remarkable observation and plenty knowledge about the sounds and music, he recognised "the presence of audible notes that were not being produced by the violin. He concluded that these notes must be generated within the ear, and he

started using them regularly in his compositions. Essentially, he would have the ear “fill in” to create a sensation of more sound than the violin was producing, the Tartini tones as they called it.[64,65]"

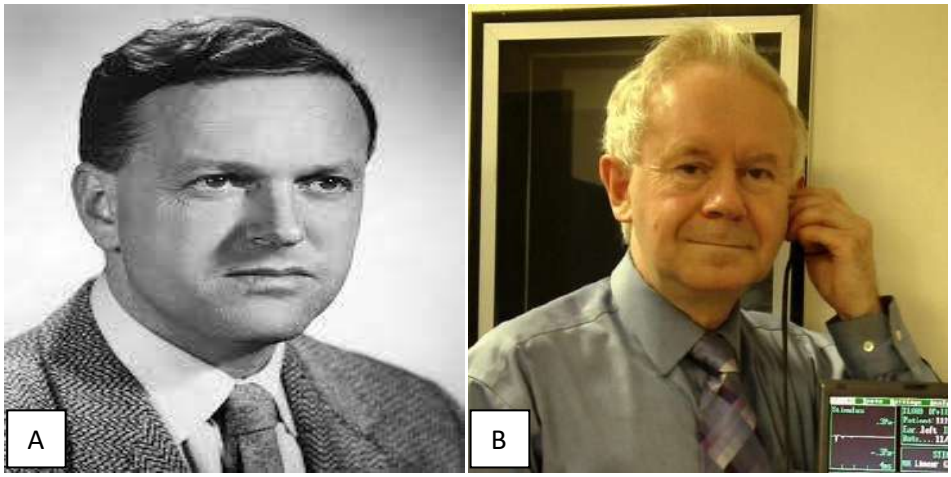
Giuseppe Tartini

The concept of Otoacoustic emissions was first forecast by a renowned Austrian-born American astrophysicist **Thomas Gold** in the year 1948.[66] He gave the concept of the “physical processes in passive cochlea” in his study.

He quoted

“It is shown that the assumption of a “passive” cochlea, where elements are brought into mechanical oscillation solely by means of the incident sound, is not tenable. The degree of resonance of the elements of the cochlea can be measured, and the results are not compatible with the very heavy damping which must arise from the viscosity of the liquid.

For this reason the “regeneration hypothesis” is put forward, and it is suggested that an electromechanical action takes place whereby a supply of electrical energy is employed to counter act the damping. (Gold, 1948) The interposition of a feedback stage . . . makes construction possible where the nerve ending abstracts much energy from a mechanical resonator. (Gold, 1948).”



A) Thomas Gold, the British physicist who in the late 1940s recognized the nonlinear nature of the cochlea.
B) David Kemp, the British physicist who discovered otoacoustic emissions.

In 1991 David Kemp bombarded the humankind with his remarkable contribution in use of Otoacoustic Emission testing (OAE) as a screening tool in newborns.[15] Due to the legendary and paradigm-shifting efforts and contributions of David T. Kemp in audiology he was bestowed with the Academy's 2019 International Award for Hearing. His exemplary work on Otoacoustic emissions (OAEs) and its discovery in 1978 unfolded the cochlear function of generation of sound. Due to the concepts put forward by David Kemp, OAE began to be used as an empirical tool for hearing screening. Considering the benefits of OAE and its implications in hearing screening, several renowned practitioners and audiologists have put forward their researches.[15]

IX) ROLE OF BRAIN STEM EVOKED RESPONSE (BERA) TEST IN NEONATAL SCREENING:

Auditory brainstem evoked response (BERA) or auditory brainstem response (ABR) is an audiological test widely used for the screening of neonates over the past 15 years.[11] This test a reliable test with good predictive value. The limitations of this test as a screening test are the cost, time and technical issues.

Jean L. Johnson et al conducted a two staged OAE and BERA protocol for screening of permanent hearing loss (PHL) in newborns.[67] The primary objective of their study was to determine whether a substantial number of infants who fail the initial OAE and pass the A-ABR (automated auditory brainstem response) have PHL at 9 months of age or not. They screened 86,634 infants at various sites. Of those infants who failed the OAE but passed the A-ABR in at least 1 ear, 1524 were enrolled in the study. They concluded that

- The newborns must be screened at an early age,
- It is crucial to counsel the family members.
- To use specific instruments and programs for newborn hearing screening especially the ones that target on identifying the mild and unilateral hearing losses also.

OAE and BERA are such equipments suggested.

- OAE involved the use of the sound pressure level of the monitored test stimulus in the ear canal and hence the intensity level of the evoking signal (clicks or tones) could be held constant for each infant regardless of differences in ear canal size.
- A-ABR on contrary in this study used by all sites delivered a 35-dB nHL click stimulus, the actual sound pressure level present at the eardrum probably varied significantly from infant to infant depending on the physical volume of the closed ear canal.
- The role of merits and demerits of 2 stage OAE/A-ABR protocol for newborn screening.

Stevens et al proposed that the efficiency of neonatal screening could be improved by using OAE testing as the primary screen, with failure referred for ABR screening. This variable could explain why some infants with mild degrees of hearing loss (and greater) may pass A-ABR screening while failing OAE.[68,69] 723 candidates were taken in his study and tested using Evoked OAE and BERA in intensive care unit. The EOAE test conducted was found to be a more practical and

quicker test. The sensitivity and specificity of the of EOAE calculated in comparison to BERA was found to be 93% and 84% respectively.

Anand S et al. conducted a prospective study on newborns delivered in Chirayu Medical College & Hospital, Bhopal. After taking informed consent, A total no. of 1052 newborn babies (with or without risk factor) were screened using a two staged OAE (between 24-72hrs) and BERA Protocol (for those who fail OAE).[70] Out of the 1052 screened,254 newborns were found to have risk factors and 798 newborns were without any risk factor. 7.8% of high risk babies (20 out of 254) and 1.12% of no high risk babies (9 out of 798) were found to have hearing loss. The total hearing impairment incidence was found to be 2.75 % (29 out of total).

Similar study was conducted by Rughani Sudhir et al in 306 newborns showed 100 high risk newborns (22%) with hearing impairment. In comparison to the non high risk category 206 (1.46%) newborns were found to have hearing loss. With the overall incidence of 8.2 % for hearing impairement.[71]

Bhatt J et al compared the sensitivity and specificity of OAE and BERA in newborn screening and stated that the incidence of permanent congenital hearing loss was 5% (5 out of 100) and in well nursing babies was found to be 0.5 % (10 times more than the high risk group).[72] They calculated the sensitivity and specificity of the Otoacoustic emissions test which was found to be as follows:

Sensitivity and specificity of OAE

Age	Sensitivity	Specificity
0 month	70 %	61 %
3 months	70 %	99 %
6 months	90 %	99 %

Another study conducted by Nagapoornima et al primarily focussed on determining the weighted incidence of hearing impairment in a standardized population of at risk and not at risk neonates seeking care at a tertiary level hospital in India.[53] Out of the 1769 cases screened 1490 candidates were not at risk and 279 were at risk. This nonrandomised prospective study was conducted at St John's medical College hospital from 1st September 2002 to 31st March 2006 using the transient OAE. In this study the incidence of hearing impairment was 3 per 279 in at risk infants screened and 7 per 1490 in not at risk infants screened. The weighted incidence in a standardized population of neonates seeking care at tertiary level centre in India is 5.60 per 1000 as per this study.

Heinemann et al conducted a study to find the cost effectiveness of newborn hearing with different instruments and found that two step screening (first with OAE and then with BERA) to be the most cost effective technique.[73]

A non-randomized, prospective study was held from August 2008 to August 2011 by Mishra G et al. All infants underwent a sequence of oto-acoustic emission (OAE) and final confirmatory auditory brainstem evoked response (ABR) audiometry. Out of 1,101 children, 1,069 children passed the test while 12 children had impaired hearing after being confirmed by ABR. Positive predictive value of OAE after multiple test increased to 100 %. This study concluded that OAE-BERA is an effective study for the neonatal screening at an early stage.[74]

Raghuwanshi SK et al [4] in their study in the ENT department of LN medical college and JK hospital Bhopal (M.P) between 2015 and 2017, screened 1250 newborns for hearing loss. Otoacoustic emissions test (Interacoustics OAE DK-5610 assens) was used as screening tool and BERA (Interacoustics BERA model-eclipse) used as the diagnostic entity.

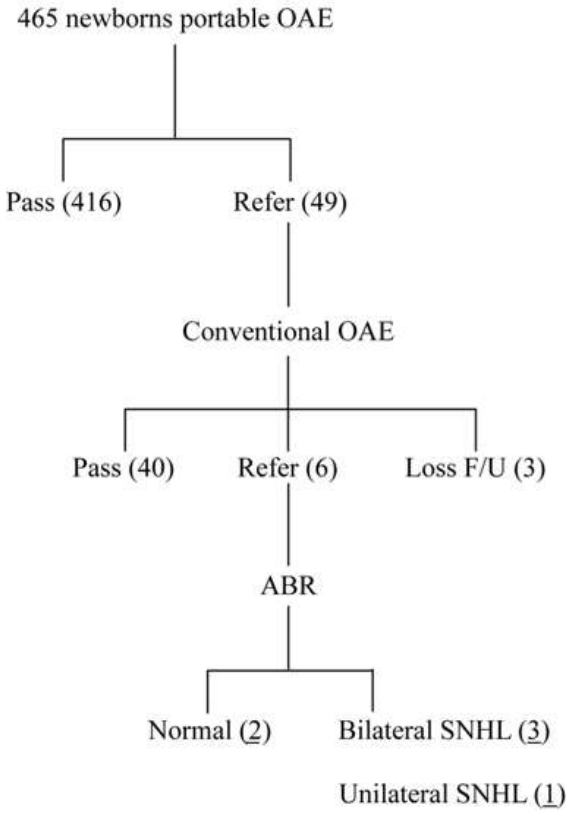
A two stage protocol was used in screening followed by BERA in those with results as refer in OAE.

The results were concluded as follows: 1250 newborns (659 boys and 591 girls) were screened. The sensitivity of OAE was found to be 66.7% and 98.8% specificity in the diagnosis of neonatal hearing impairment. Its positive and negative predictive value was 33.3% and 99.7% respectively. They concluded that OAE is a good screening test for hearing loss in neonates but the results must be confirmed with BERA.

Keren et al.[75] in their study concluded that since ‘early identification and intervention of a hearing disorder leads to better language skills, lower educational costs and increased lifetime productivity, the cost-effectiveness of universal neonatal hearing screening is much better than no screening or selective screening.’

Apuzzo and Yoshinaga-Itano [55] found that "infants who are identified with hearing loss early have a distinct advantage over their later identified peers" and "identification and intervention begun before 2-1/2 years benefits all infants with hearing loss, regardless of hearing impairment. This benefit is especially evident for subjects who were identified by two months of age." [76]

In another study by Khaimook W et al at Songklanagarind Hospital conducted in the year 2004-2006 screened the high risk neonates according to the Joint Committee for hearing loss with an objective of screening the neonates for determining the prevalence and risk factor of neonatal hearing loss in the high-risk group. The results were concluded as follows:[77]



They stated Otoacoustic emissions was a very quick and noninvasive technique and suitable for hearing screening in infants.

In another study by Abdullah A et al all infants born in the hospital were screened for hearing loss with a portable otoacoustic emission (OAE) before discharge. At the age of two months, a second OAE test was repeated on newborns who failed the initial test. Those who failed the second test were re-tested at three months of age. When these infants failed the third OAE test, a brainstem evoked response (BERA) test was performed. During the study period, 4,219 infants were born in the hospital, and 3,762 (89.2 percent) underwent OAE screening. 620 (19.7 percent) of them failed the first screening test, and 506 (81.6 percent) of them came for a second stage-screening test. In the third stage screening at three months of age, only 39 (65 percent) patients turned up. Of these, ten infants passed the OAE test and 29 failed. However, when these infants underwent BERA, 13 had normal BERA and 16 have abnormal BERA. The prevalence of hearing loss in this study was 0.42 percent (16/3,762).[51]

Out of 153 neonates screened by Di Stadio A et al ,100 babies (73.9%) presented normal hearing at TEOAE and A-ABR. 15 (9.8%) had a REFER response at TEOAE and a PASS response at A-ABR. 25 (16.3%) had a REFER A-ABR response and were addressed to clinical ABR evaluation. All children with altered A-ABR results underwent clinical ABR. The results of clinical ABR showed that 12/25 (48%) children had a moderate (41 to 55 dB HL) and profound (>91 dB HL) SNHL; 11/25 (44%) were false positives and had a normal hearing. Among children with hearing loss, 4 (33.4%) had a unilateral profound SNHL, 3 (25%) a bilateral profound SNHL, 3 (25%) a unilateral moderate SNHL, 1 (8.3%) a bilateral moderate SNHL and 1 (8.3%) received a diagnosis of auditory neuropathy. 2 children were lost during follow up.[78]

In a study by Kamath R, 425 babies were screened initially by OAE. Out of these 405(95.2%) gave results as PASS and 20 neonates (4.7%) gave results as REFER. These 20 neonates were subjected to second screening by OAE. In which 15(75%) neonates gave PASS results and 5(25%) had results as REFER. Dropout after second screening was 1. Total number of babies on whom BERA was performed were 4(0.94%) and only 1 neonate was found to have hearing loss.[79]

MATERIAL AND METHODS

Source of data

This was a prospective observational study conducted in the level III Neonatal intensive care unit (NICU) of a tertiary care teaching hospital, Dayanand Medical College and Hospital, Ludhiana by the department of Otorhinolaryngology. The study prospectively included neonates (<28 days of age) admitted in NICU with high risk factors.

Method of collection of data

It was a prospective observational study conducted over a period of 1 year from 1st April 2021 to 31st March 2022. The study was approved by the Institutional ethics committee. Written informed consent was obtained from the parents/legal guardians prior to incorporation into the study.

All admissions in NICU were evaluated and neonates having the high risk factors (as per the inclusion criteria) were enrolled in the study.

Inclusion criteria

1. Family history of hereditary childhood sensorineural hearing loss
2. In utero infections (Toxoplasmosis, Rubella, Cytomegalovirus, Herpes simplex virus infections and Syphilis)
3. Craniofacial anomalies
4. Low birth weight <2500 g
5. Hyperbilirubinemia (at serum levels requiring exchange transfusion)
6. Ototoxic medications
7. Bacterial meningitis
8. Perinatal asphyxia (APGAR of 5 at 1 minute or of 6 at 5 minutes)
9. Mechanical ventilation lasting 5 days or longer
10. Stigmata or other findings associated with a syndrome known to include a sensorineural hearing loss

Exclusion criteria

1. Neonates with congenital external ear malformation precluding the use of OAE as a screening tool.
2. Neonates with non consenting parents.

The following data was compiled and OAE and BERA (if required) was performed as documented in the proforma.

- Demographic details
- Clinical symptoms with detailed antenatal and birth history
- Presence of high risk factor
- Vital signs and examination findings
- Laboratory investigations with respect to the high risk factor present
- Results of OAE testing
- Results of BERA testing (If performed)
- Results of the screening
- Provisional diagnosis
- Treatment administered

The high risk neonates were screened for hearing loss using Otoacoustic Emissions (OAE), as a screening tool, in two stages. In the OAE Stage I, testing using OAE was conducted prior to discharge. The interpretation of the test was in the form of REFER or PASS and documented in the file. The neonates in whom OAE results were 'REFER' were called for a repeat OAE (Stage II) after 6 weeks (usually coinciding with their visit in paediatrics unit for vaccination). Those neonates in whom results were 'PASS' were considered normal and were therefore not followed up. Neonates in whom results were still 'REFER' after Stage II OAE underwent a BERA test to determine the presence/absence of hearing loss. Counselling of parents regarding the rehabilitation of hearing was done for those neonates in whom OAE and BERA results showed hearing impairment.

STUDY DURATION:

1 year from 1st April 2021 to 31st March 2022

OUTCOME: Screening of high risk neonates for hearing loss.

Method of collection of audiological data:

The DPOAE test was conducted for all the neonates enrolled in the study in the ENT OPD using the Neurosoft non portable device connected to the computer in our audiology room.

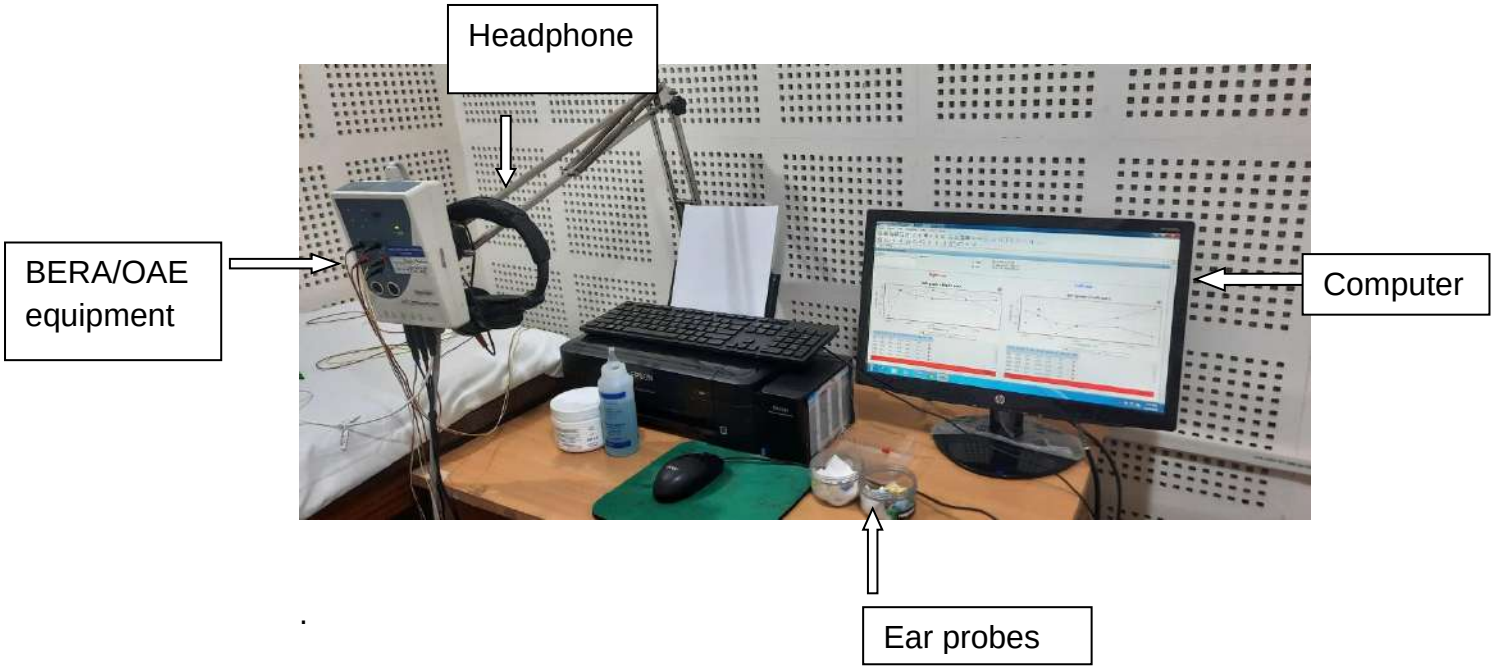
The earplugs were placed in both ears of the newborn (one ear at a time) during sleep or at the time of end of feeding. Once the earplug was placed in the ear, the DPOAE were measured using the primary tones labelled as f1 and f2 at a frequency ranging from 500 Hz to 8000Hz. The duration of this test was around 2-5 mins per ear across the frequency range and the results were recorded on the DP gram which was displayed on the computer. The results were documented as PASS or REFER.

In the neonates with overall test results as REFER, BERA was performed.

Before performing BERA test, it was ensured that the newborn's testing area was cleaned with no application of oil/moisturiser/medicine over the testing area.

The screening using BERA was done in our audiology room in quiet environment. The electrodes were placed at the various sites as follows: ground electrode at the forehead, non-inverting electrode at vertex and inverting electrode over the mastoid prominence or ear lobe. The headphones were then applied to give the sound stimulus. The intensity was increased gradually from 40dB to 100 dB. The auditory stimulus was given in the form of clicks and tone bursts and the hearing sensitivity measured for 1k-4k Hz. The waveforms were observed at the computer, and we analysed the absolute latency of waves I,III,V, interpeak latencies(I-III,III-V,I-V), interaural difference of interpeak I-V.

The BERA/OAE apparatus used in our OPD

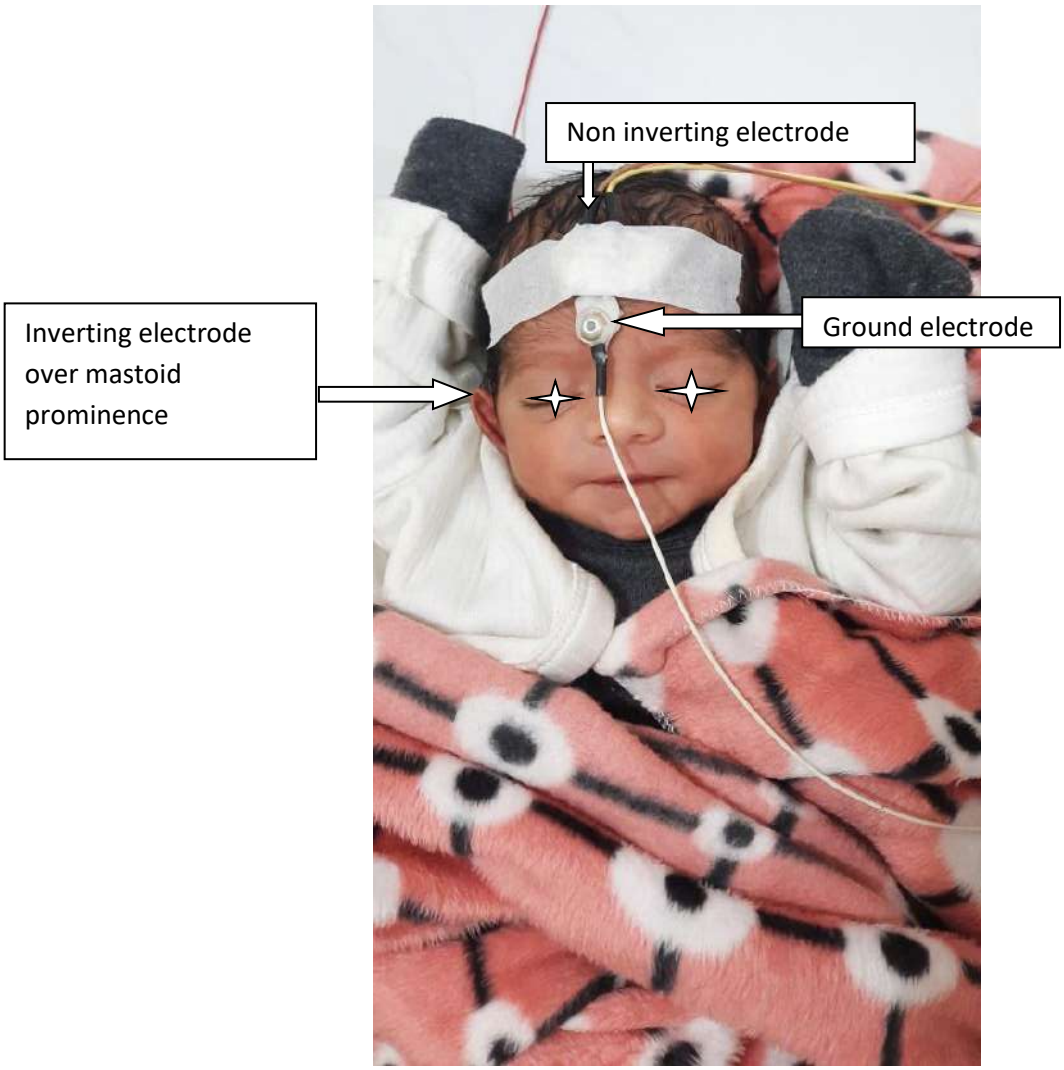


.

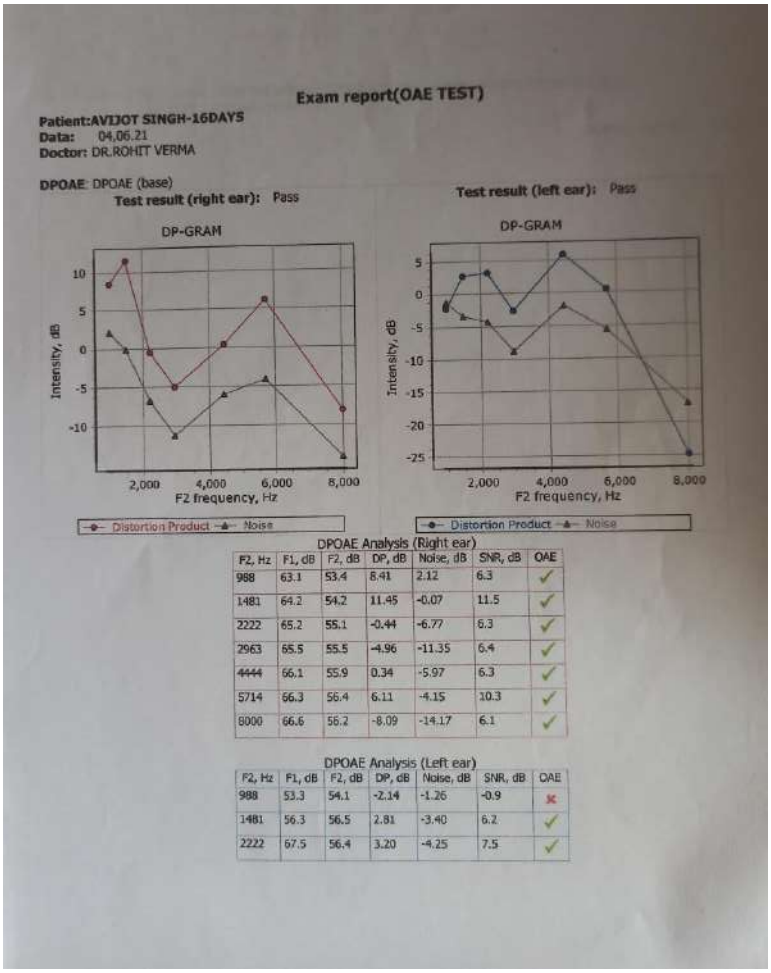
The setup of OAE/BERA Test

Ear probes of different sizes used in OAE

OAE test being performed in right ear



The positioning of electrodes in BERA test while the neonate is sleeping. (Before application of headphones.)



Exam report(OAE TEST)

Patient:AVIJOT SINGH-16DAYS
Data: 04.06.21
Doctor: DR.ROHIT VERMA

DPOAE: DPOAE (base)

Test result (right ear): Pass

Test result (left ear): Pass

DP-GRAM

Intensity, dB

F2 frequency, Hz

Distortion Product Noise

DPOAE Analysis (Right ear)

F2, Hz	F1, dB	F2, dB	DP, dB	Noise, dB	SNR, dB	OAE
988	63.1	53.4	8.41	2.12	6.3	✓
1481	64.2	54.2	11.45	-0.07	11.5	✓
2222	65.2	55.1	-0.44	-6.77	6.3	✓
2963	65.5	55.5	-4.96	-11.35	6.4	✓
4444	66.1	55.9	0.34	-5.97	6.3	✓
5714	66.3	56.4	6.11	-4.15	10.3	✓
8000	66.6	56.2	-8.09	-14.17	6.1	✓

DPOAE Analysis (Left ear)

F2, Hz	F1, dB	F2, dB	DP, dB	Noise, dB	SNR, dB	OAE
988	53.3	54.1	-2.14	-1.26	-0.9	✗
1481	56.3	56.5	2.81	-3.40	6.2	✓
2222	67.5	56.4	3.20	-4.25	7.5	✓

Result of OAE Test

Statistical analysis:

The incidence of hearing loss was analysed from the data of OAE and BERA.

All the clinico demographic factors were studied and correlations of hearing loss in high risk neonates were made to various positive risk factors. Continuous data variables were presented as means and standard deviations (SD). Categorical data were presented as percentages and Chi Square test was used to derive comparisons. For all statistical testing, p value < 0.05 was considered significant.

OBSERVATIONS & RESULTS

In our prospective observational study, 50 neonates were included.

DEMOGRAPHIC PROFILE

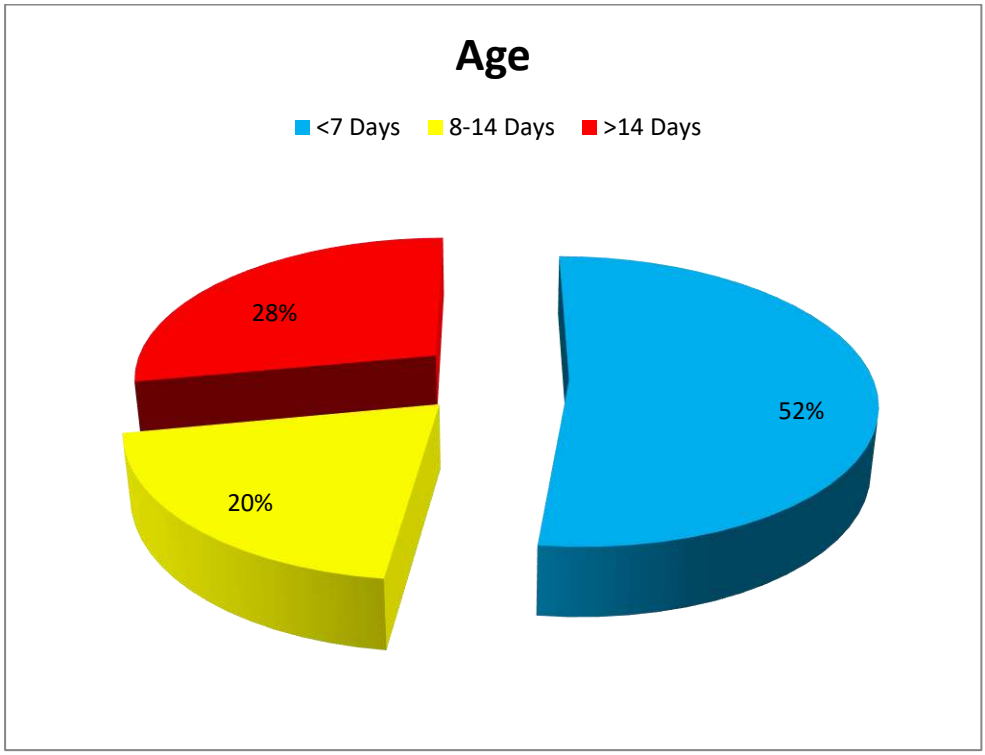
I) AGE OF NEONATES

Out of these 50 newborns, 26 newborns (52%) were <7 days, 10 newborns (20%) 8-14days of age and 14 newborns (28%) >14days of age. (Table 1 and figure 1)

Table1: Age of neonates

Age Group	No. of cases	Percentage
< 7 Days	26	52.0
8-14 Days	10	20.0
> 14 Days	14	28.0
Total	50	100.0

Figure 1: Age of neonates



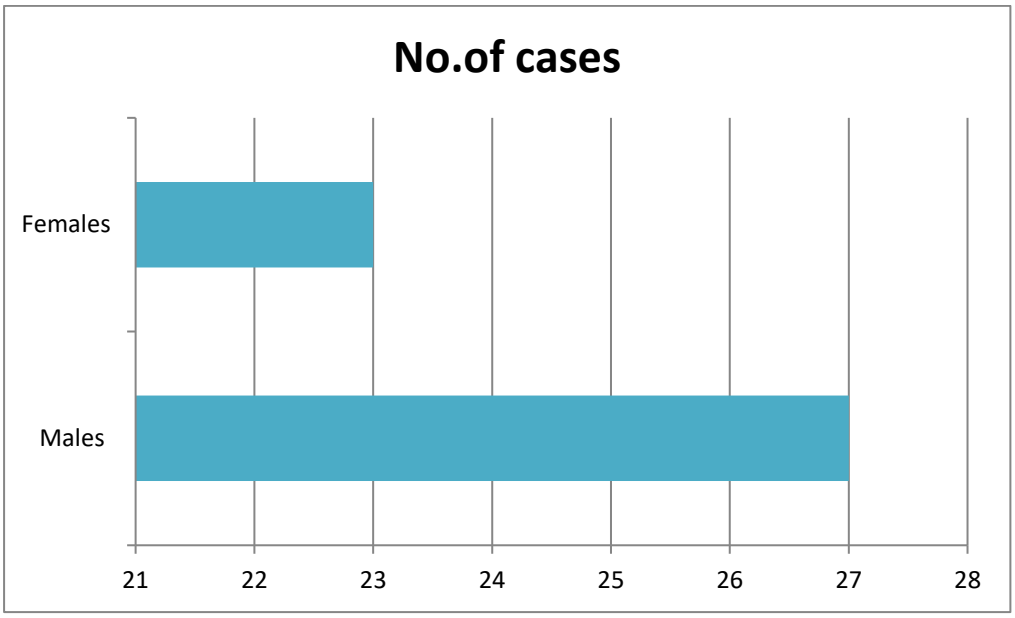
II) GENDER

Out of the 50 neonates, 23 neonates (46%) were females and 27 neonates (54%) were males.(table 2, figure 2)

Table 2: Gender of neonates

Gender	No. of cases	Percentage
Female	23	46.0
Male	27	54.0
Total	50	100.0

Figure 2: Gender distribution of neonates



III) AGE OF PARENTS

The maternal age ranged from 21years to 36 years. The age of fathers ranged from 23 years to 38 years. (Table 3, 4 /figure 3, 4)

Table 3: Maternal age (In years)

Age of Mother	No. of cases	Percentage
21	1	2.0
22	1	2.0
23	7	14.0
24	5	10.0
25	8	16.0
26	10	20.0
27	4	8.0
28	10	20.0
29	2	4.0
33	1	2.0
36	1	2.0
Total	50	100.0

Figure 3: Age of Mothers (In Years)

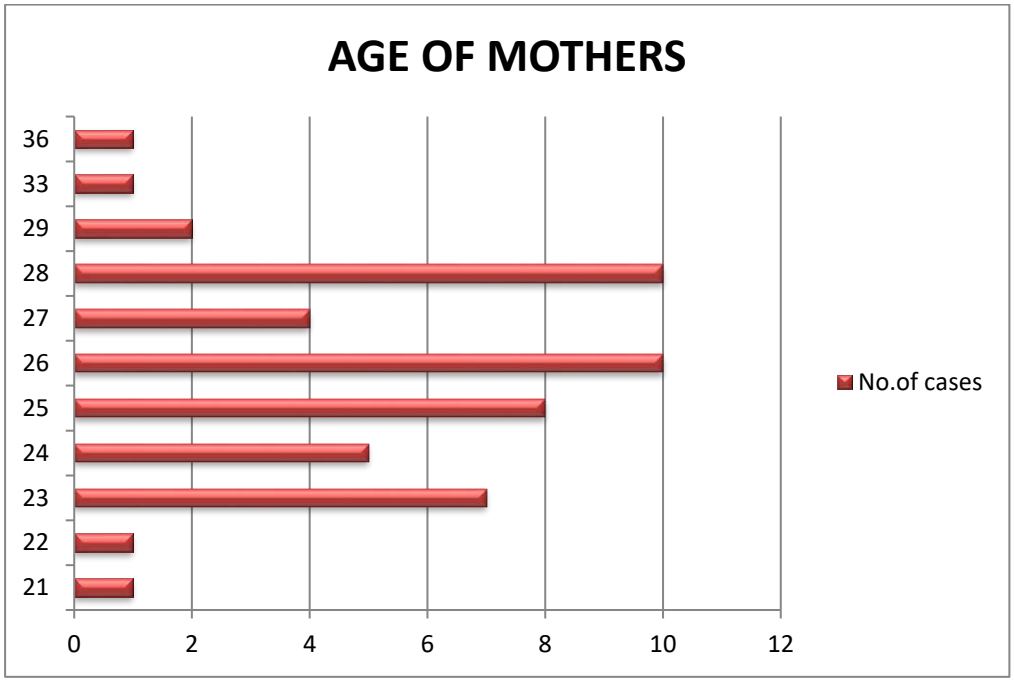
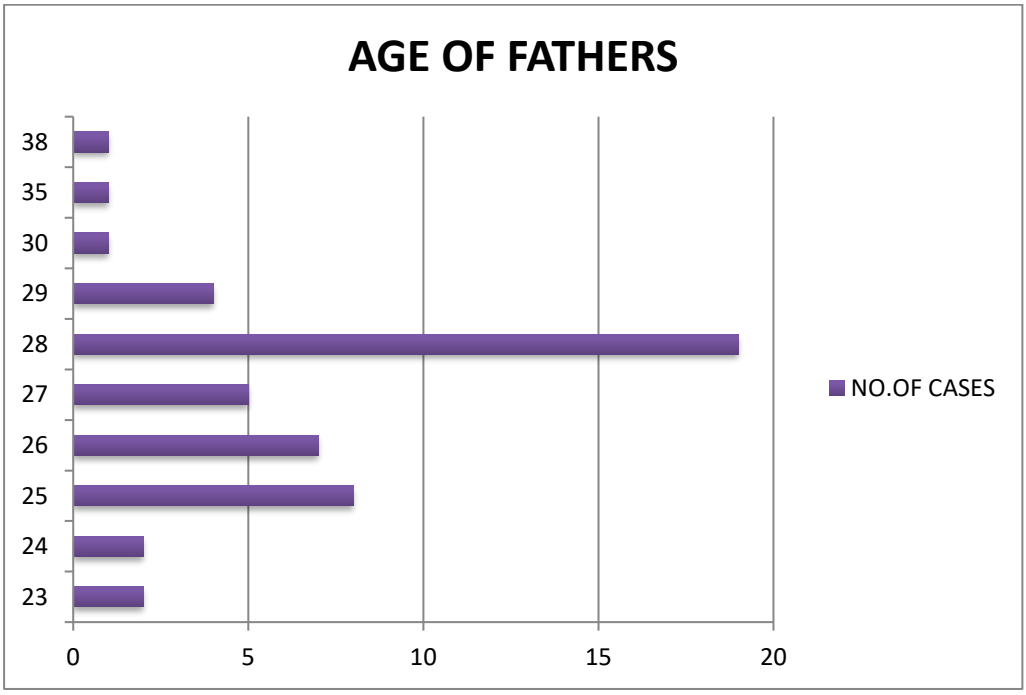


Table 4: Age of Fathers (In Years)

Age of Father	No. of cases	Percentage
23	2	4.0%
24	2	4.0%
25	8	16.0%
26	7	14.0%
27	5	10.0%
28	19	38.0%
29	4	8.0%
30	1	2.0%
35	1	2.0%
38	1	2.0%
Total	50	100.0%

Figure 4: Age of Fathers (In Years)



IV) OCCUPATION OF PARENTS

The table and figure given below show the occupation of mothers and fathers (figure 5, 6 table 5, 6)

Table 5: Occupation of Mothers

Mother occupation	No. of cases	Percentage
TEACHER	7	14.0
BAKER	1	2.0
DOCTOR	3	6.0
HOUSEHELPER	1	2.0
HOUSEWIFE	34	68.0
SERVICE	2	4.0
STUDENT IN COLLEGE	1	2.0
WORKER	1	2.0
Total	50	100.0

Figure 5: Occupation of Mothers

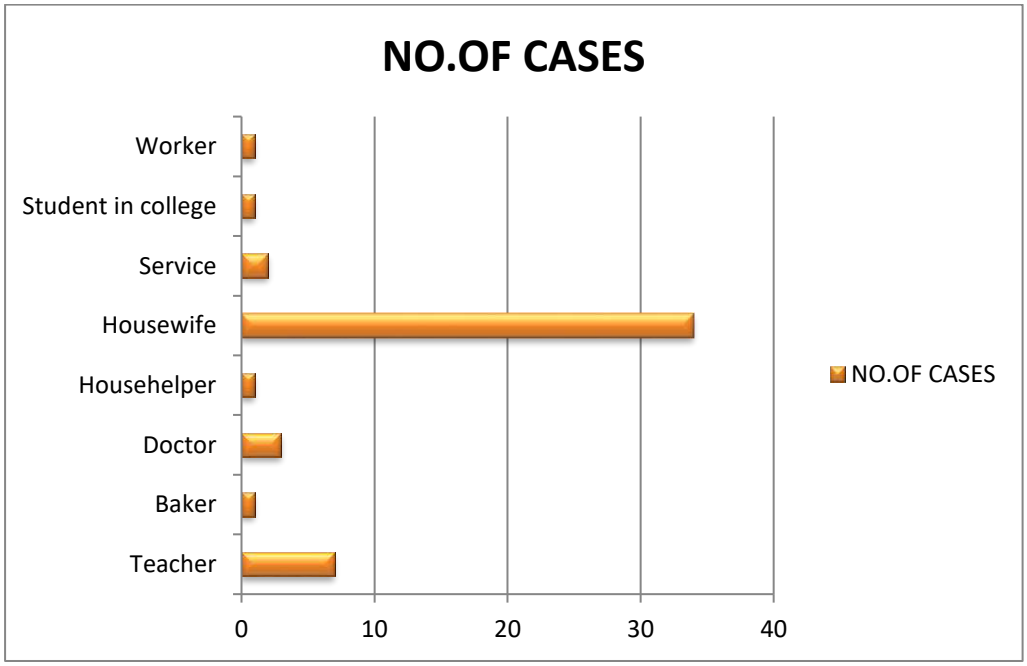
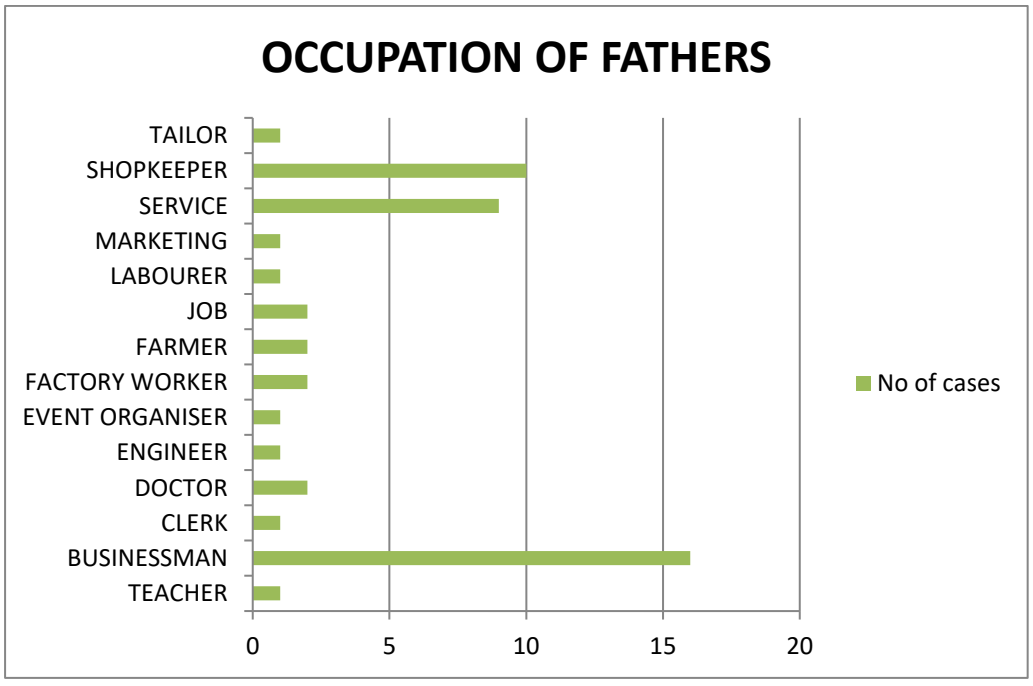


Table 6: Occupation of Fathers

Father occupation	No. of cases	Percentage
TEACHER	1	2.0%
BUSINESSMAN	16	32.0%
CLERK	1	2.0%
DOCTOR	2	4.0%
ENGINEER	1	2.0%
EVENT ORGANISER	1	2.0%
FACTORY WORKER	2	4.0%
FARMER	2	4.0%
JOB	2	4.0%
LABOURER	1	2.0%
MARKETING	1	2.0%
SERVICE	9	18.0%
SHOPKEEPER	10	20.0%
TAILOR	1	2.0%
Total	50	100.0%

Figure 6: Occupation of Fathers



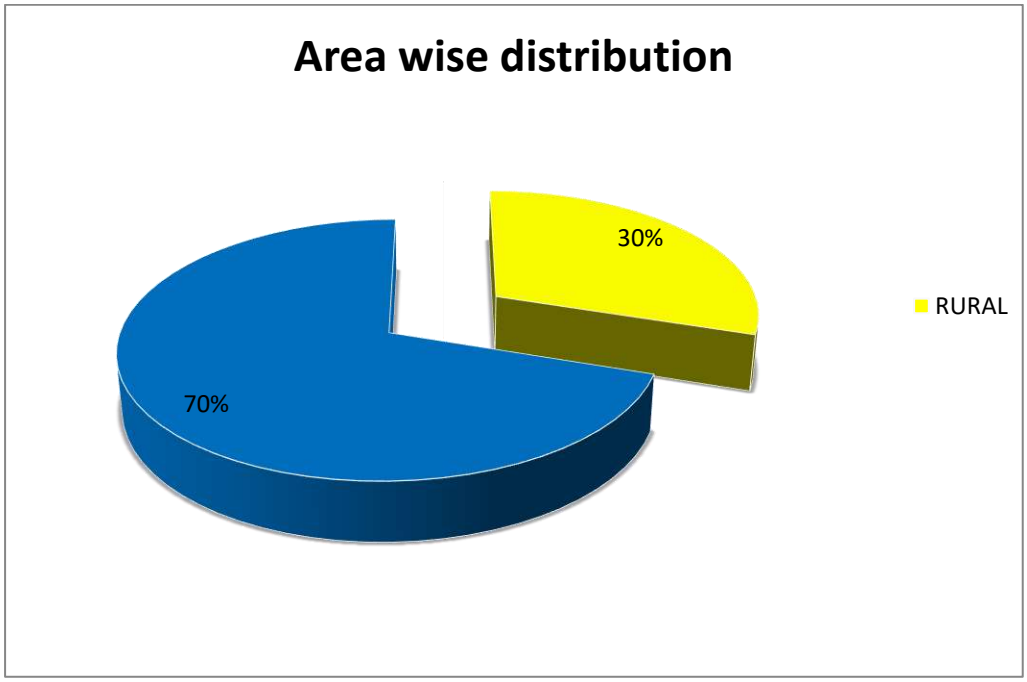
V) AREA WISE DISTRIBUTION

Out of 50 neonates, 15 neonates (30%) were from rural area and 35 (70%) from urban area (table 7, figure 7)

Table 7: Area wise distribution of neonates

Community	No. of cases	Percentage
RURAL	15	30.0
URBAN	35	70.0
Total	50	100.0

Figure 7: Areawise distribution of neonates



A) CLINICAL PROFILE

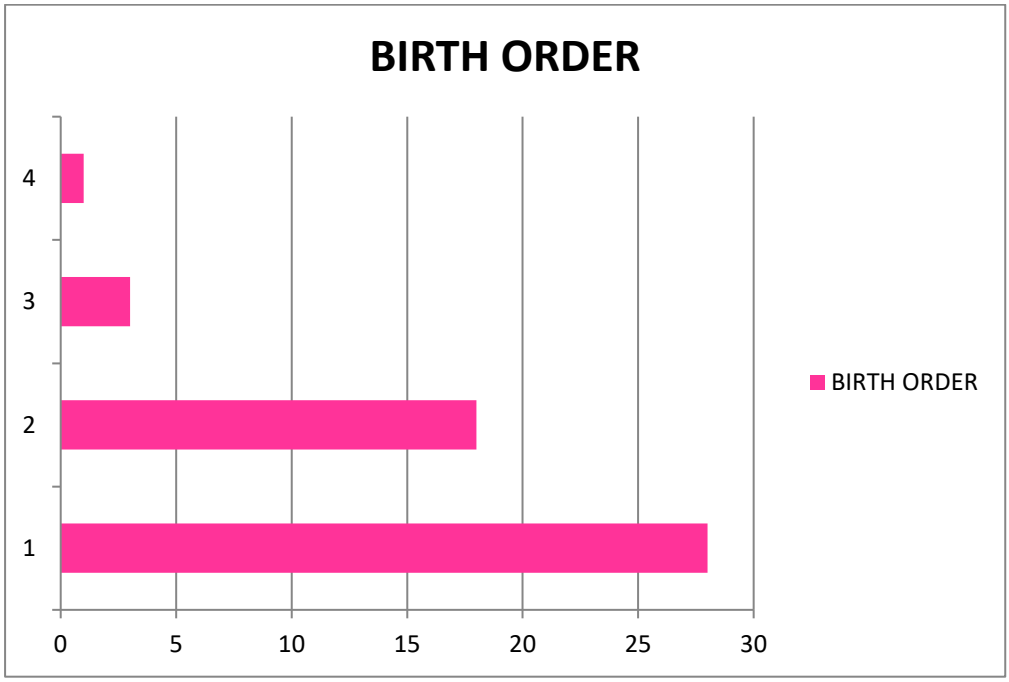
1) BIRTH ORDER

In our study population, 28 neonates (56%) had a birth order of 1, 18 (36%) of birth order 2, 3 (6%) of birth order 3 and 1(2%) of birth order 4. (table 8, figure 8)

Table 8: Birth order of neonates

Birth Order	No. of cases	Percentage
1	28	56.0%
2	18	36.0%
3	3	6.0%
4	1	2.0%
Total	50	100.0%

Figure 8: Birth order of neonates



2) APGAR Score ;

The APGAR score was observed at 1 min and 5 mins.

Out of 50 neonates, 5 (10%) had an APGAR score of 3 ,2(4%) had a score of 4, 8(16%) had a score of 5, 4 (8%) had a score of 6 , 13(26%) had a score of 7, 14(28%) had a score of 8, 1 (2%) had a score of 9 and 3 (6%) had an unknown score at 1 min (TABLE 9, FIGURE 9)

Table 9: APGAR score at 1 min

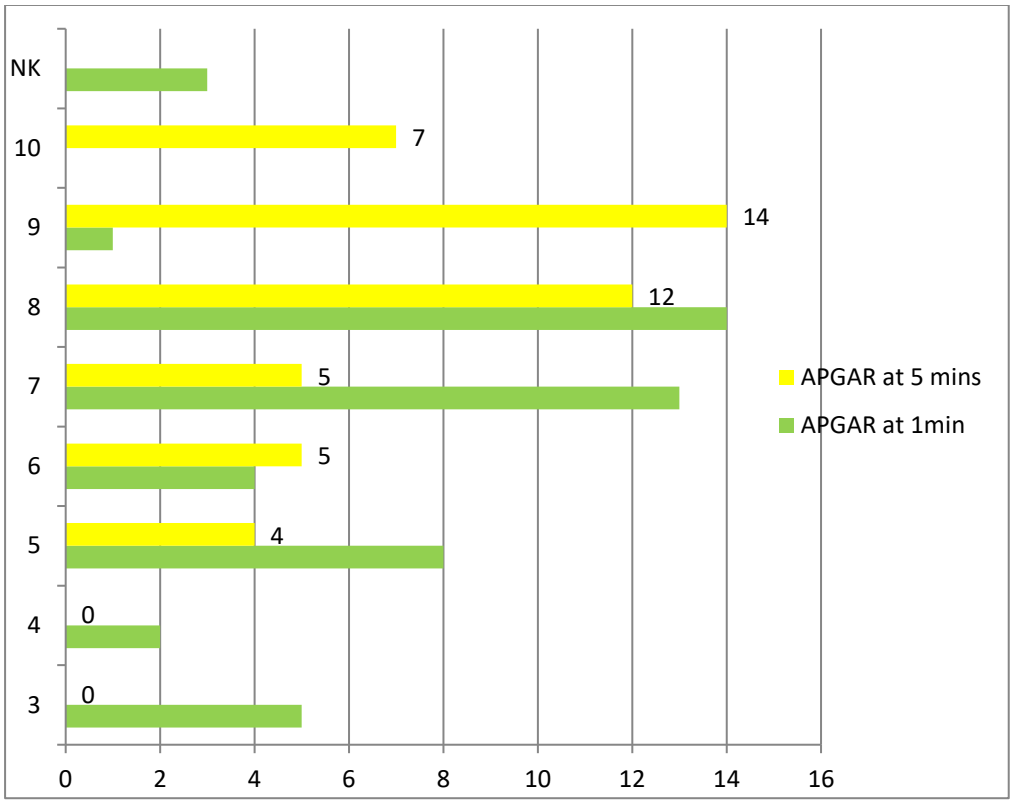
APGAR score At 1 min	No. of cases	Percentage
3	5	10.0%
4	2	4.0%
5	8	16.0%
6	4	8.0%
7	13	26.0%
8	14	28.0%
9	1	2.0%
Not known(NK)	3	6.0%
Total	50	100.0%

Out of 50 neonates, 4 (8%) had an APGAR score of 5, 5(10%) had a score of 6, 5(10%) had a score of 7, 12(24%) had a score of 8, 14(28%) had a score of 9, 7 (14%) had a score of 10 and 3(6%) had an unknown score at 5 mins. (Table 10, figure 9)

Table 10: APGAR score at 5 mins

APGAR score At 5 mins	No. of cases	Percentage
5	4	8.0%
6	5	10.0%
7	5	10.0%
8	12	24.0%
9	14	28.0%
10	7	14.0%
Not known(NK)	3	6.0%
Total	50	100.0%

Figure 9: APGAR Score at 1 min and 5 mins



3) ANTENATAL HISTORY

Parameters considered while taking the antenatal history of mothers were history of presence of hypertension, preeclampsia, diabetes thyroid disorder, epilepsy, sensorineural hearing loss and history of drug intake. (figure 10)

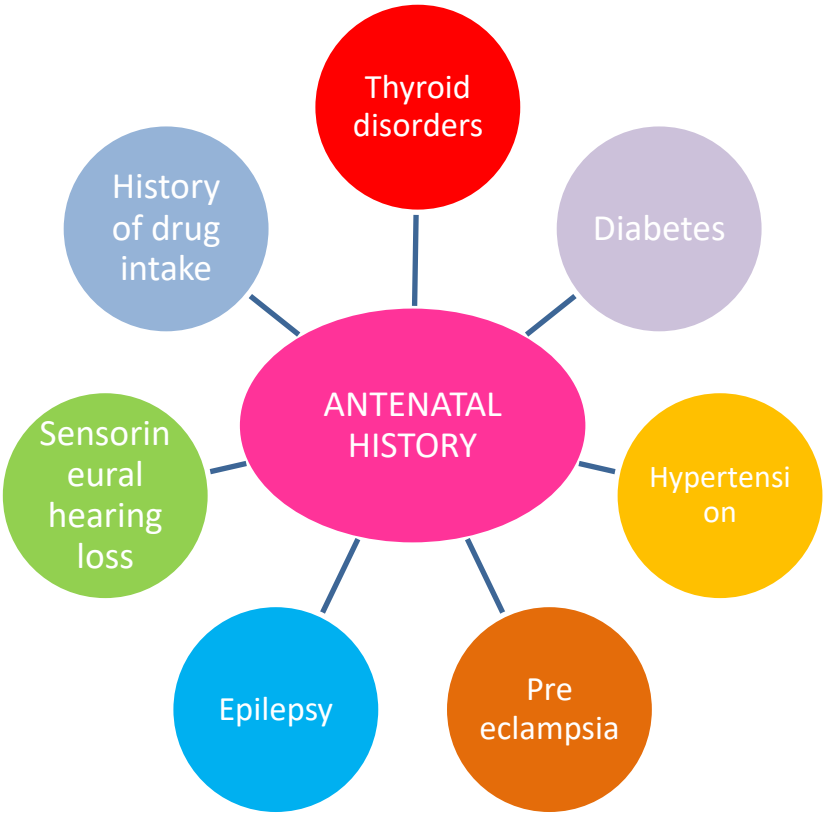


Figure 10: Antenatal Parameters

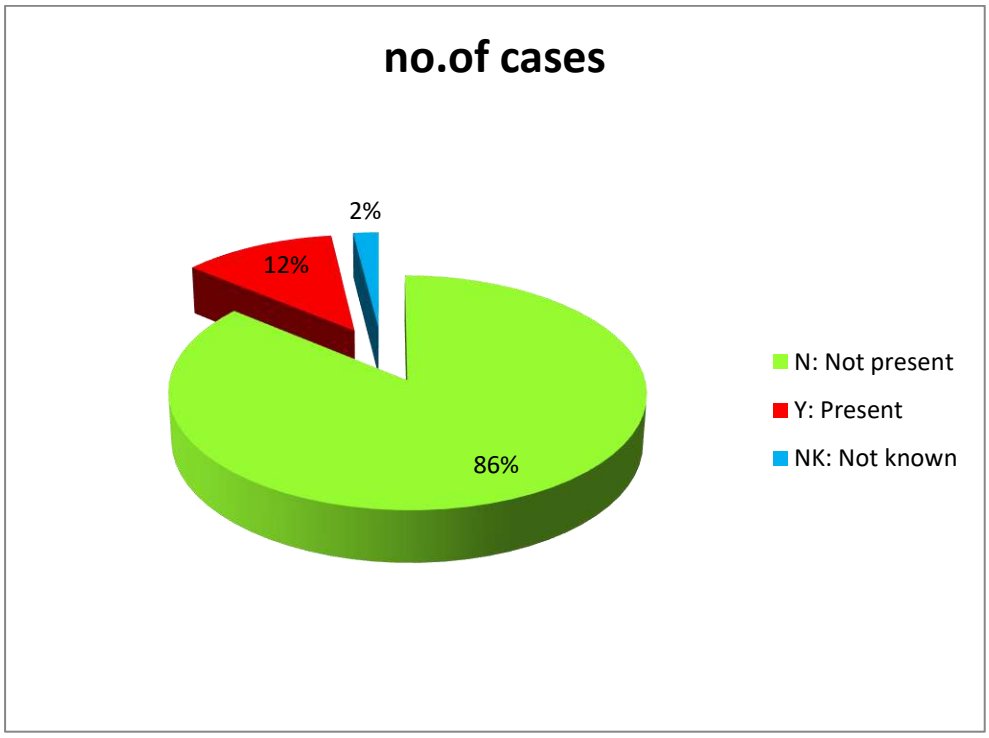
a) History of hypertension :

43 mothers (86%) of our study population had no positive antenatal history of hypertension, 6(12%) had a positive history and 1 (2%) mother had an unknown status. (Table 11, figure 11)

Table 11: Antenatal history of hypertension

H/O Hypertension	No. of cases	Percentage
No	43	86.0%
Not known	1	2.0%
Yes	6	12.0%
Total	50	100.0%

Figure 11: Antenatal history of hypertension



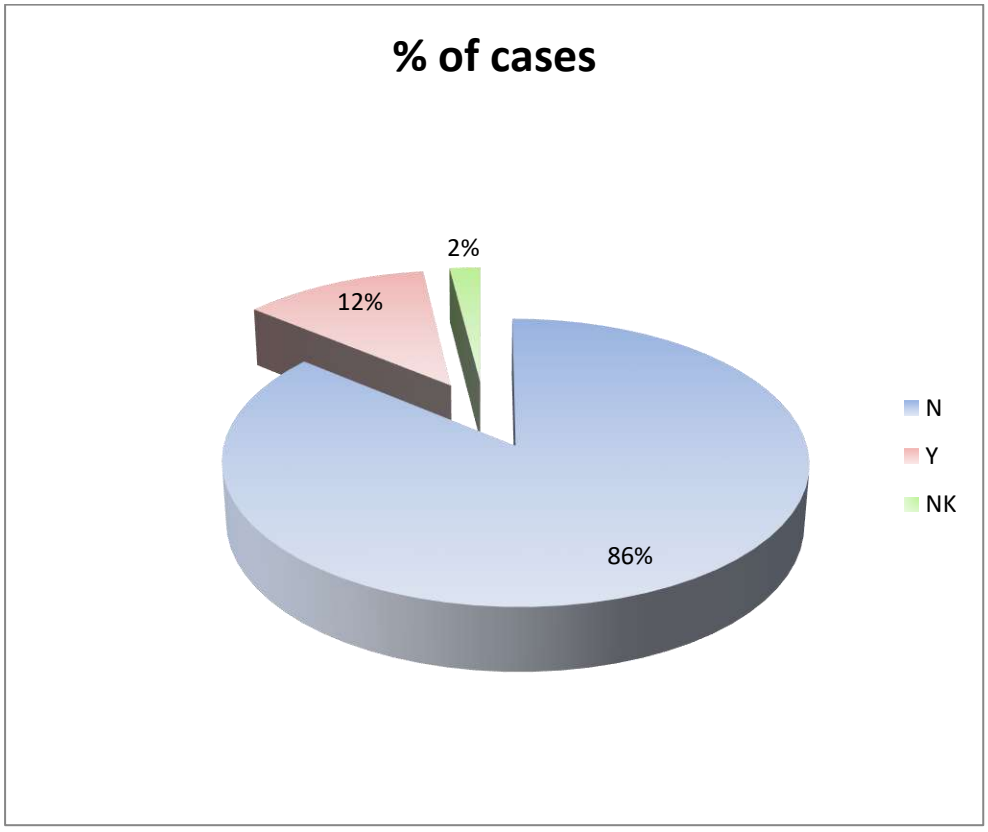
b) Antenatal history of preeclampsia :

43 mothers (86%) of our study population had no positive antenatal history of preeclampsia, 6(12%) had a positive history and 1 (2%) mother had an unknown status. (Table 12, figure 12)

Table 12: Antenatal history of preeclampsia

H/O Preeclampsia	No. of cases	Percentage
No	43	86.0%
Not known	1	2.0%
Yes	6	12.0%
Total	50	100.0%

Figure 12: Antenatal history of preeclampsia



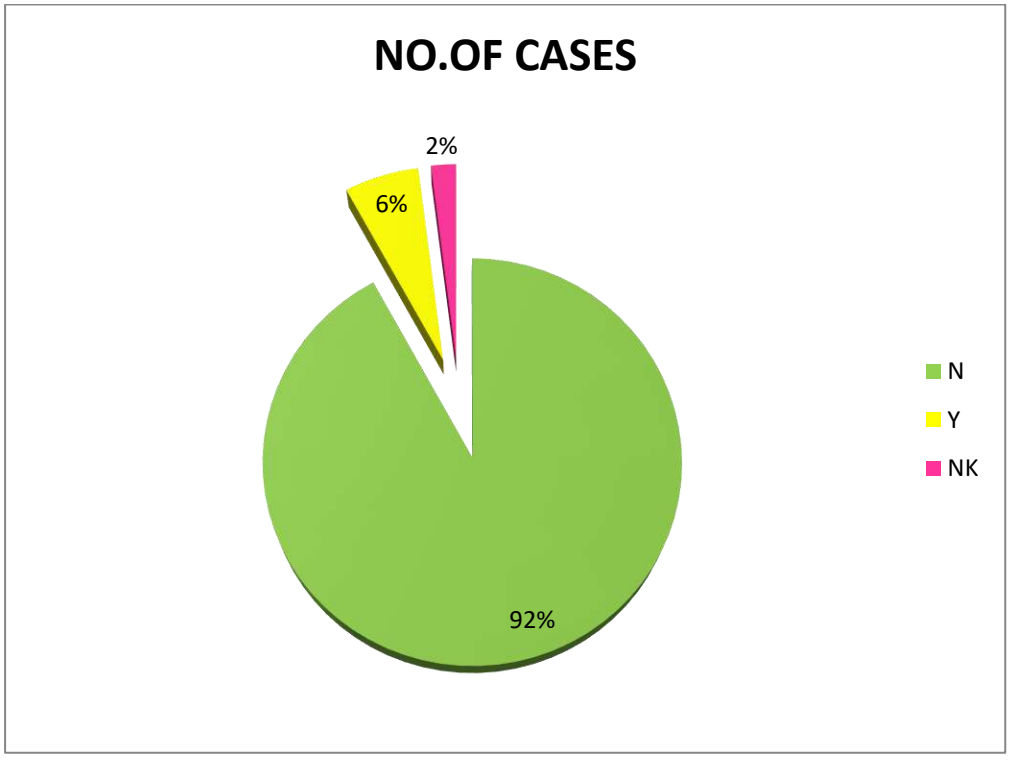
c) Antenatal history of diabetes:

Out of 50 mothers 46(92%) mothers had a negative antenatal history of diabetes, 3(6%) had a positive history and 1(2%) had an unknown diabetes status. (Table: 13)

Table 13: Antenatal history of diabetes

h/o Diabetes	No. of cases	Percentage
No	46	92.0%
Not known	1	2.0%
Yes	3	6.0%
Total	50	100.0%

Figure 13: Antenatal history of diabetes



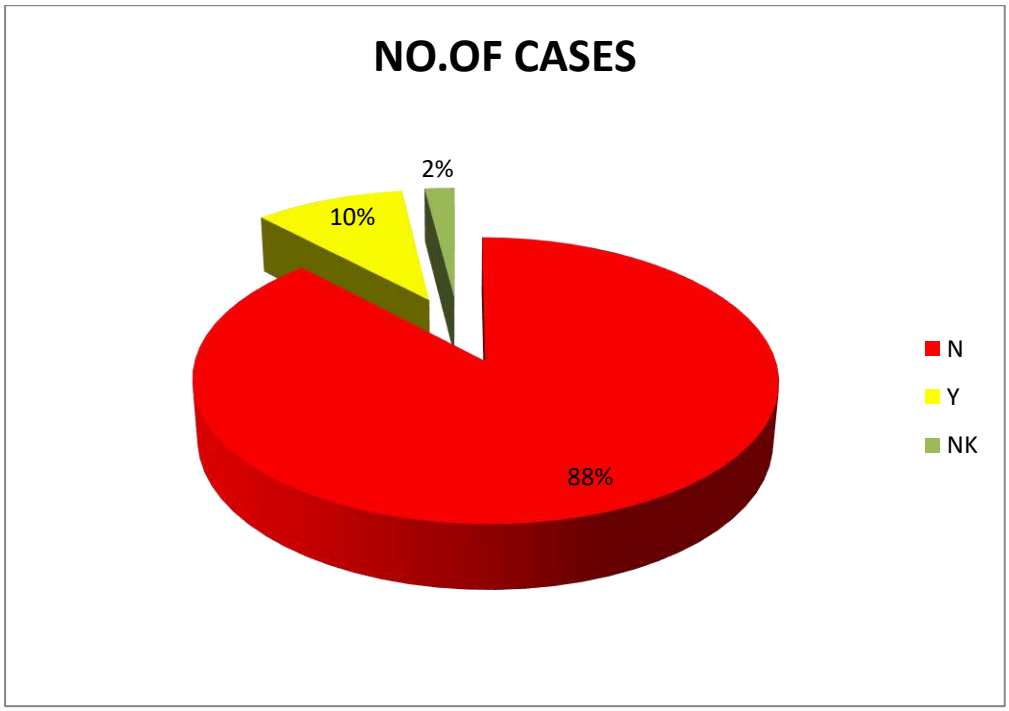
d) Antenatal history of thyroid disorders:

Out of 50 mothers, 5(10%) neonates had a positive antenatal history of thyroid disorders, 44(88%) had a negative history and 1(2%) had an unknown status. (Table 14, Figure 14)

Table 14: Antenatal history of thyroid disorders

H/o thyroid disorders	No. of cases	Percentage
No	44	88.0%
Not known	1	2.0%
Yes (OVERT)	5	10.0%
Total	50	100.0%

Figure 14: Antenatal history of thyroid disorders



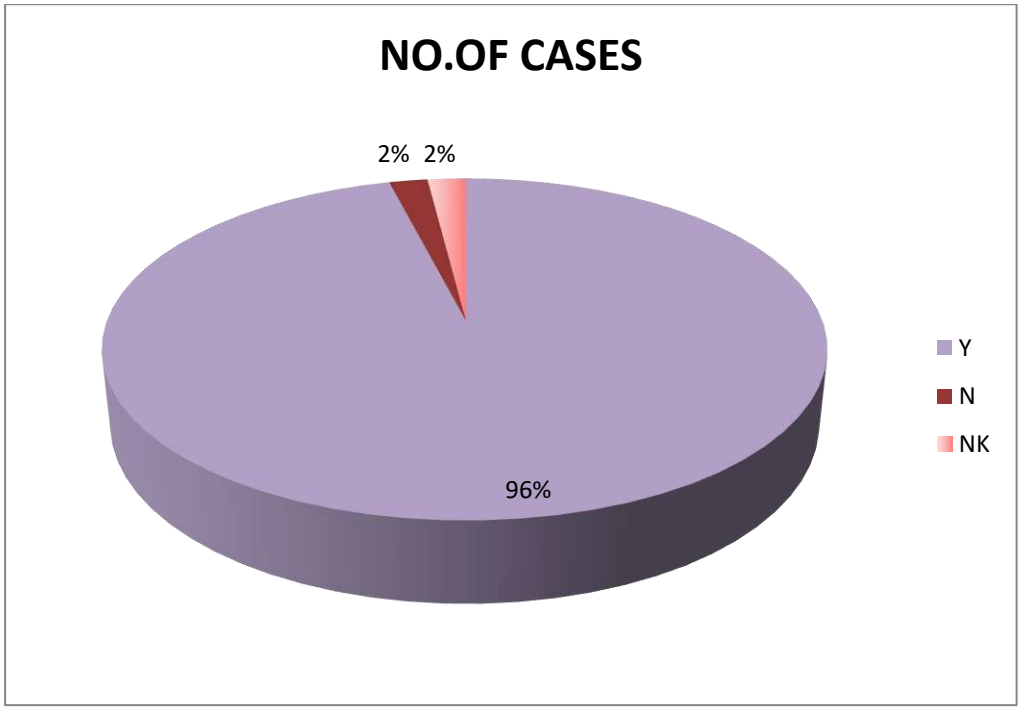
e) Antenatal history of Epilepsy:

Out of 50 mothers, 1 (2%) had a positive history of epilepsy, 48(96%) had a negative history and 1(2%) had an unknown status. (figure 15, table 15)

Table 15: Antenatal history of epilepsy

H/o Epilepsy	No. of cases	Percentage
No	48	96.0%
Not known	1	2.0%
Yes	1	2.0%
Total	50	100.0%

Figure 15: Antenatal history of epilepsy



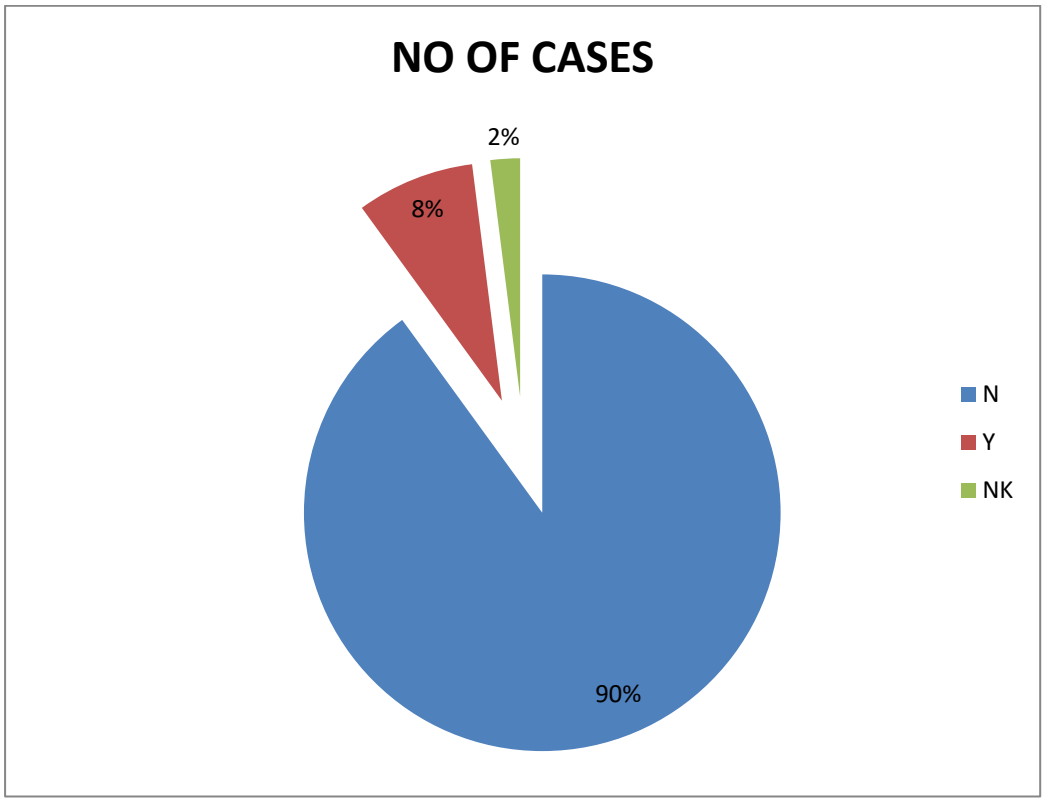
f) History of sensorineural hearing loss during antenatal period:

4 mothers (8%) had a history of hearing loss during the antenatal period, 45 (90%) had no history, while 1 (2%) had an unknown status. (Table 16, Figure 16)

Table 16: Antenatal history of SNHL

H/o Sensorineural hearing loss	No. of cases	Percentage
No	45	90.0%
Not known	1	2.0%
Yes	4	8.0%
Total	50	100.0%

Figure 16: Antenatal history of SNHL



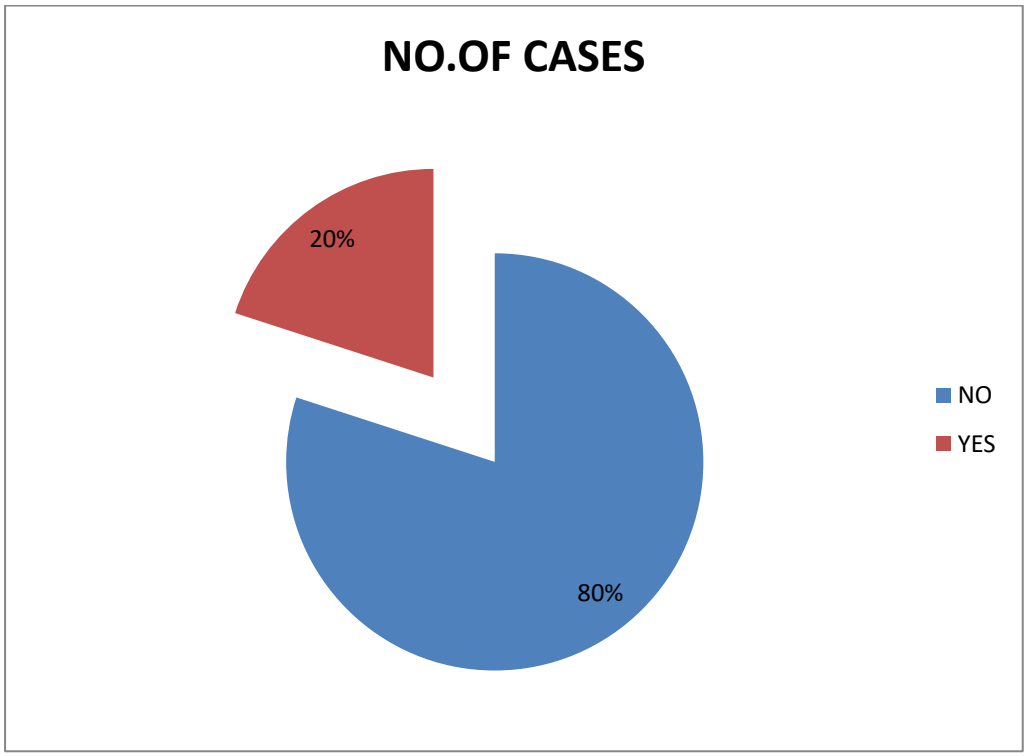
g) Maternal history of drug intake :

20% of mothers (10 mothers) had a positive history of drug intake whereas 80% (40 mothers) had no history.(Table 17,figure 17)

Table 17: Maternal history of drug intake

H/o Maternal drug intake	No. of cases	Percentage
NO	40	80.0%
YES	10	20.0%
Total	50	100.0%

Figure 17: Maternal history of drug intake



4) BIRTH HISTORY

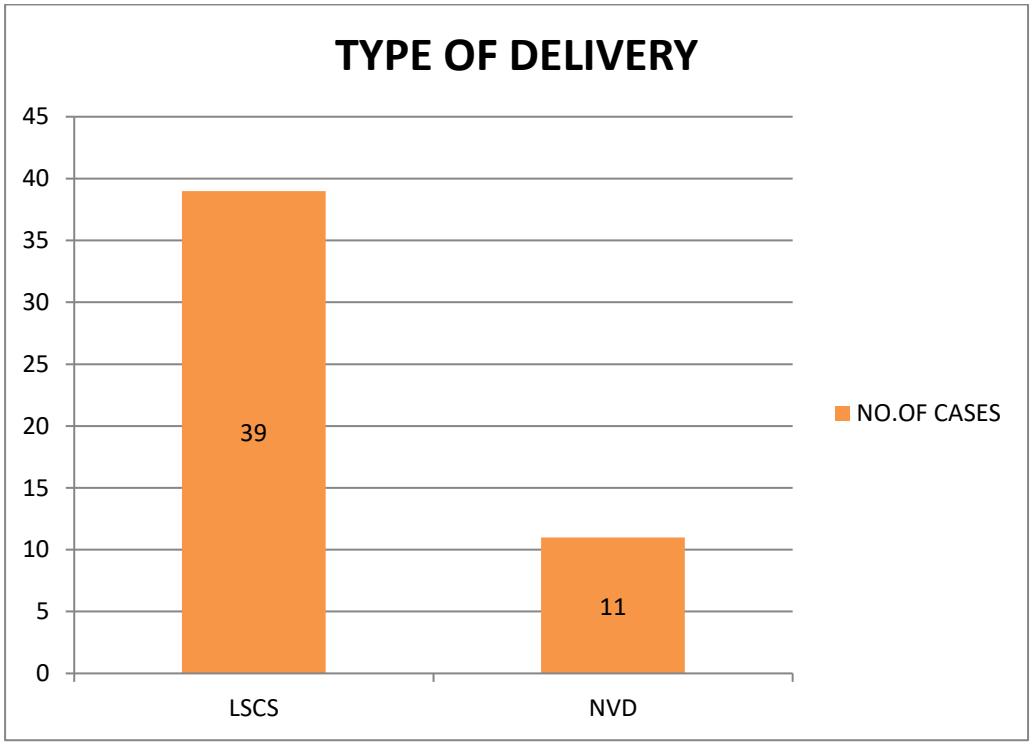
Under this category we studied the type of delivery and place of delivery in our study population.

A) TYPE OF DELIVERY: 39 (78%) Of neonates are born with lower segment caesarean section (LSCS) and 11 (22%) with normal vaginal delivery (NVD). (Table 18,Figure18)

Table 18: Type of Delivery

Type of Delivery	No. of cases	Percentage
LSCS	39	78.0%
NVD	11	22.0%
Total	50	100.0%

Figure 18: Type of Delivery

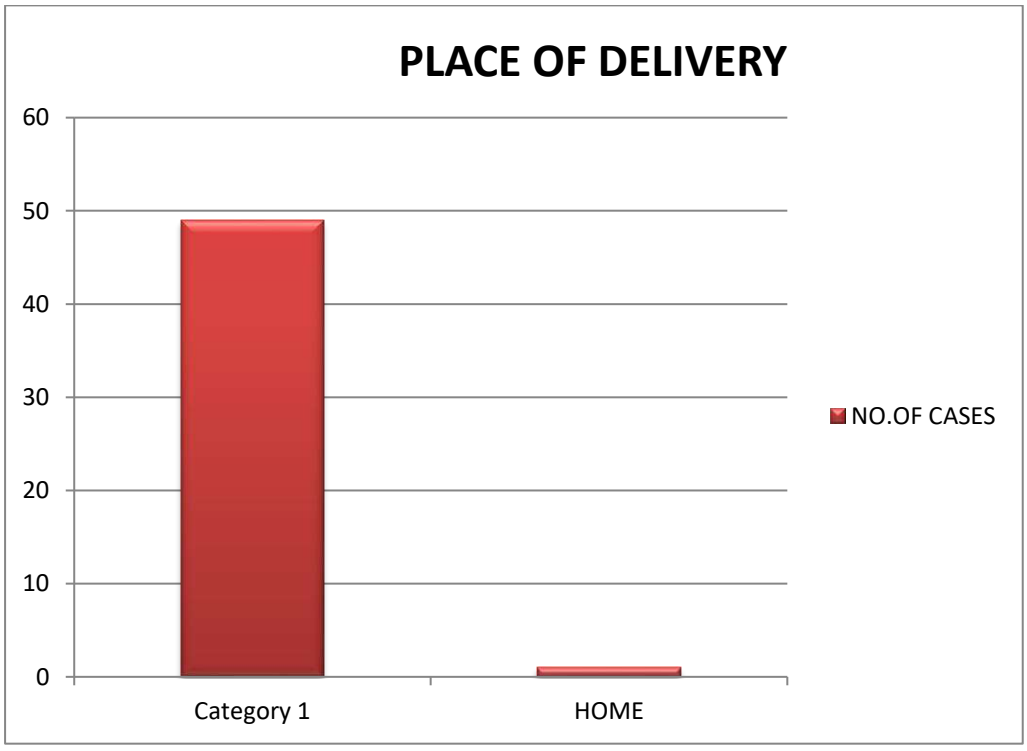


B) PLACE OF DELIVERY : 49 (98%) of the neonates were born in hospital and 1 neonate (2%) was born at home via normal vaginal delivery (Table 19, Figure 19)

Table 19: Place of Delivery

Place of Delivery	No. of cases	Percentage
HOME	1	2.0%
HOSPITAL	49	98.0%
Total	50	100.0%

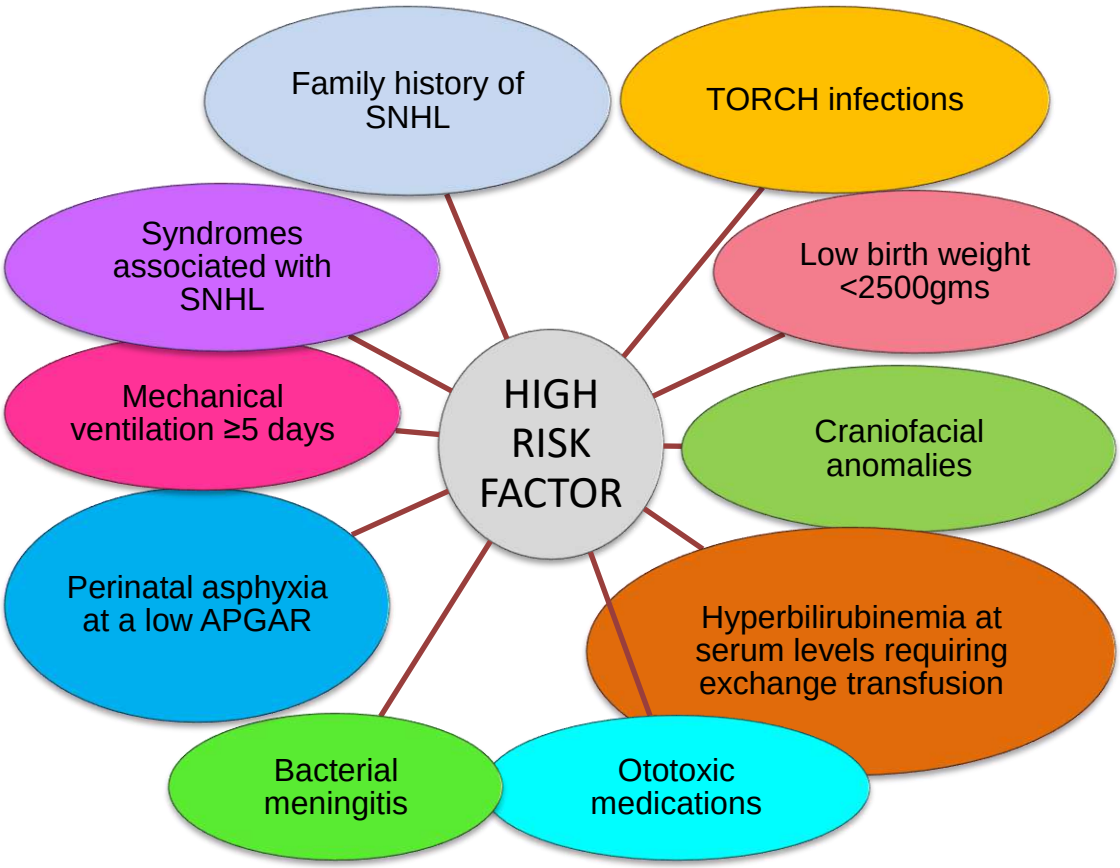
Figure 19: Place of Delivery



5) HIGH RISK FACTORS

The following risk factors were taken in our study.(figure 20)

Figure 20: High risk factors



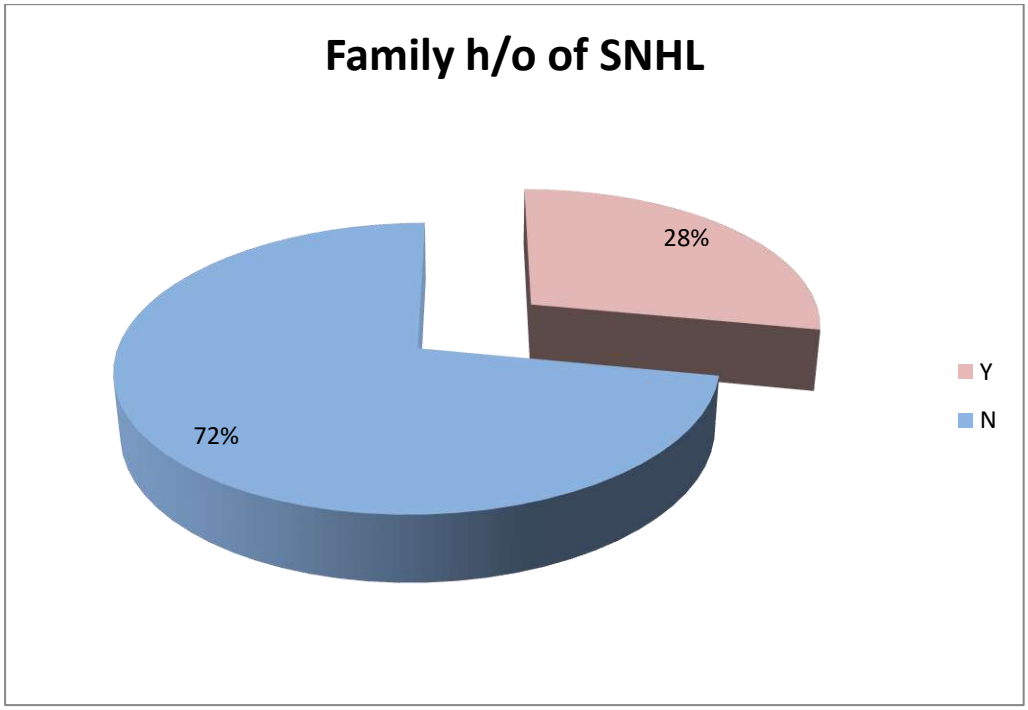
I.Family history of SNHL

Out of 50 neonates, 14(28%) had a family history of SNHL and 36 (72%) did not. (Table 20, figure 21)

Table 20: Distribution of family history of SNHL in neonates

Family history of SNHL	No. of cases	Percentage
NO	36	72.0%
YES	14	28.0%
Total	50	100.0%

Figure 21: Distribution of family history of SNHL in neonates



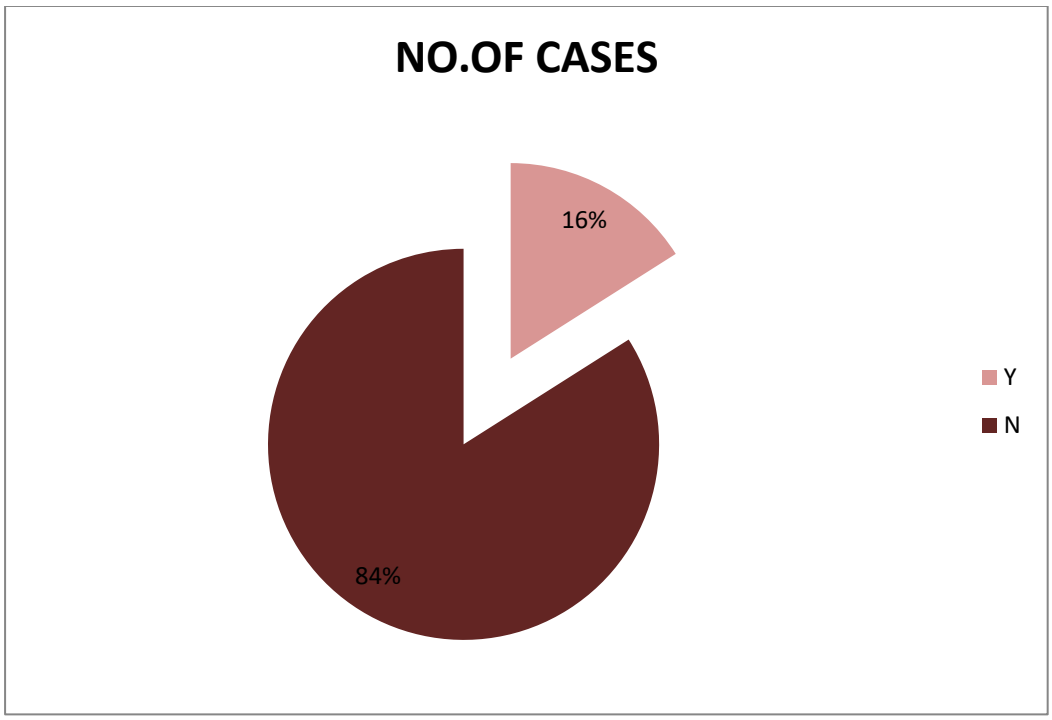
II) TORCH infections:

The presence of toxoplasmosis, rubella, cytomegalovirus, herpes simplex, HIV and other neonatal infections were seen in the newborns. 42 neonates (84% of the total) had no history of TORCH infections in the postnatal period whereas 8 neonates (16%) had a positive history of TORCH infections. (Table 21, figure 22)

Table 21: Distribution of history of TORCH infections in neonates

TORCH Infections	No. of cases	Percentage
No	42	84.0%
Yes	8	16.0%
Total	50	100.0%

Figure 22: Distribution of history of TORCH infections in neonates



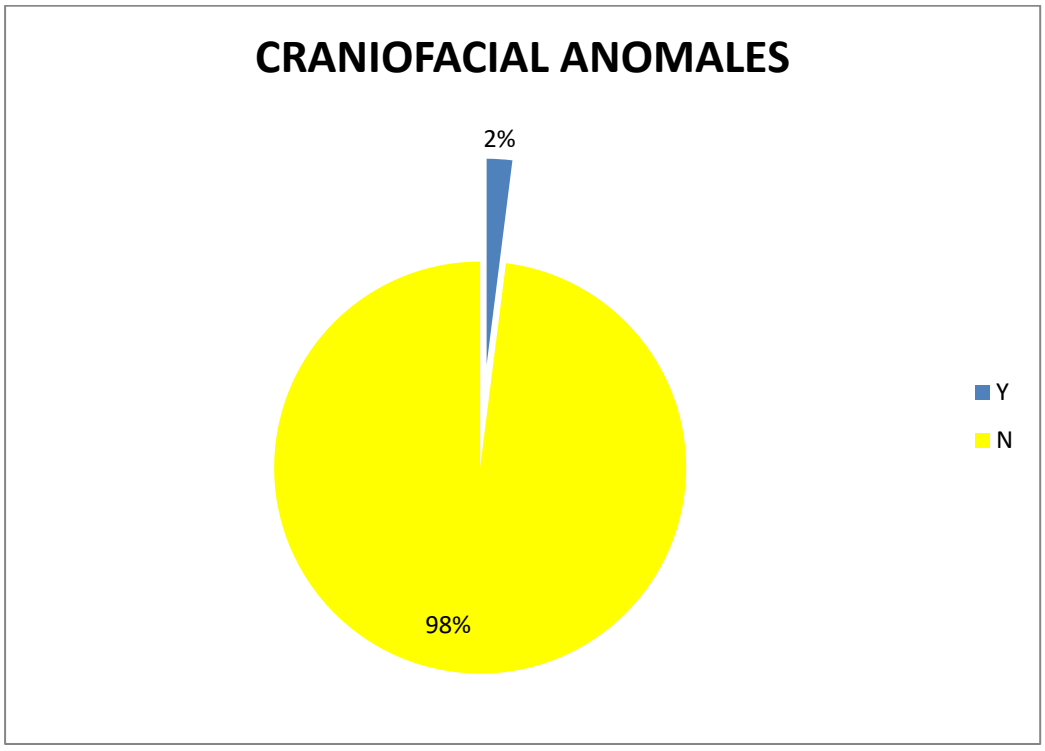
III) CRANIOFACIAL ANAMOLIES:

One neonate (2%) out of the total was found to have a craniofacial anomaly whereas 49 neonates (98%) did not have a positive history. (Table 22, figure 23)

Table 22: Distribution of craniofacial anomalies in neonates

Craniofacial anomalies	No. of cases	Percentage
NO	49	98.0%
YES	1	2.0%
Total	50	100.0%

Figure 23: Distribution of craniofacial anomalies in neonates



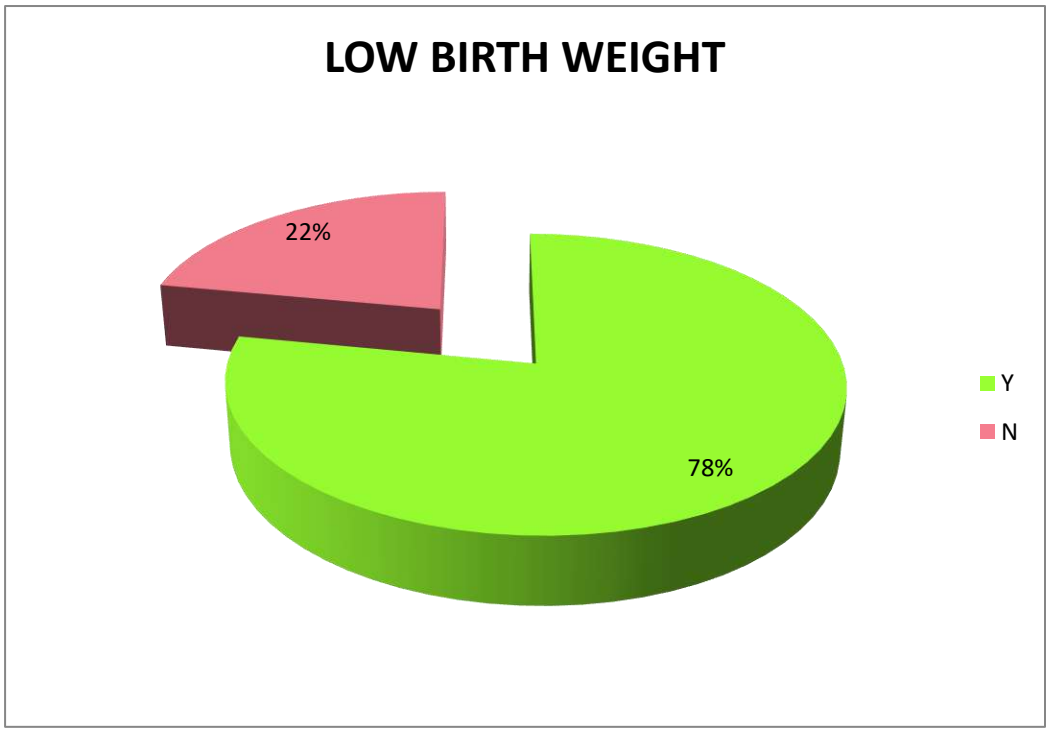
IV) LOW BIRTH WEIGHT

Out of 50 neonates screened 39 neonates (78%) had a positive history of low birth weight (<2500gms) and 11 neonates (22%) had no history of low birth weight. (Table 23, figure 24)

Table 23: Distribution of Low birth weight in neonates

Low birth weight	No. of cases	Percentage
No	11	22.0%
Yes	39	78.0%
Total	50	100.0%

Figure 24: Distribution of Low birth weight in neonates



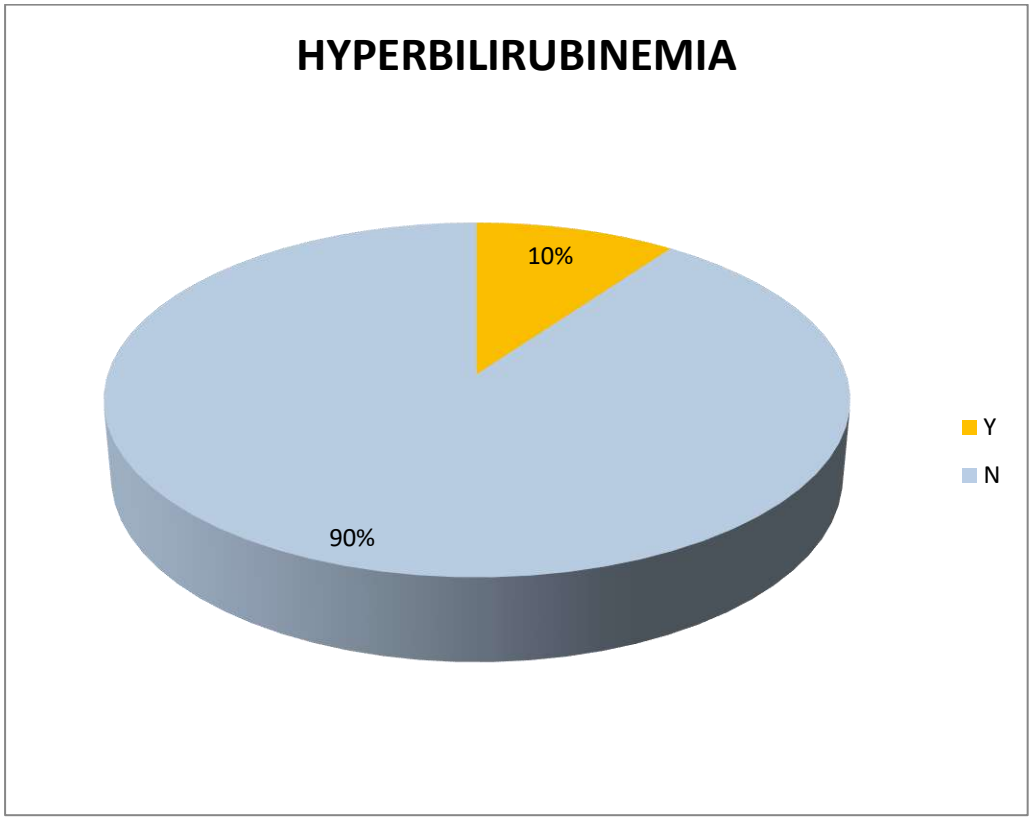
V) HYPERBILIRUBINEMIA (with serum levels requiring exchange transfusions)

Out of 50 neonates, 5 (10% of total study population) were suffering from hyperbilirubemia and 45(90%) had no such history. Table 24, figure 25)

Table 24: Distribution of hyperbilirubinemia in neonates

Hyperbilirubinemia	No. of cases	Percentage
No	45	90.0%
Yes	5	10.0%
Total	50	100.0%

Figure 25: Distribution of hyperbilirubinemia in neonates



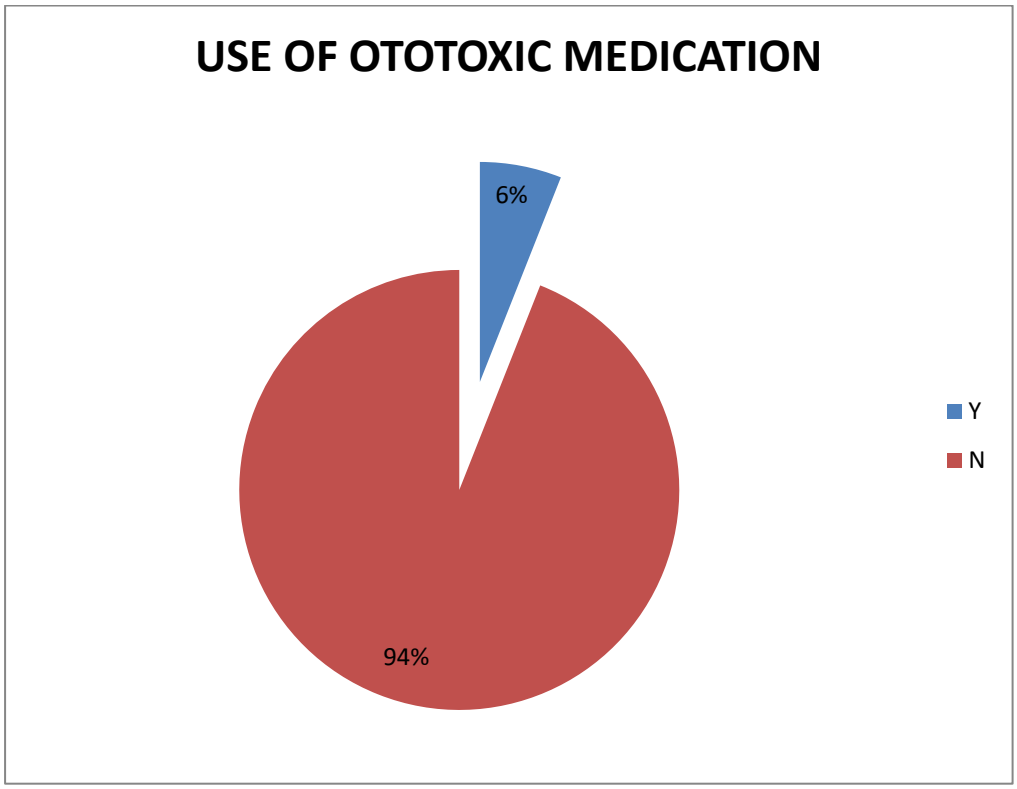
VI) OTOTOXIC MEDICATIONS

The use of ototoxic medications like amikacin was seen in 3(6%) neonates. whereas 47 neonates (94%) had no such history. (Table 25, figure 26)

Table 25: Distribution of use of ototoxic drugs in neonates

Ototoxic medications	No. of cases	Percentage
No	47	94.0%
Yes	3	6.0%
Total	50	100.0%

Figure 26: Distribution of use of ototoxic drugs in neonates



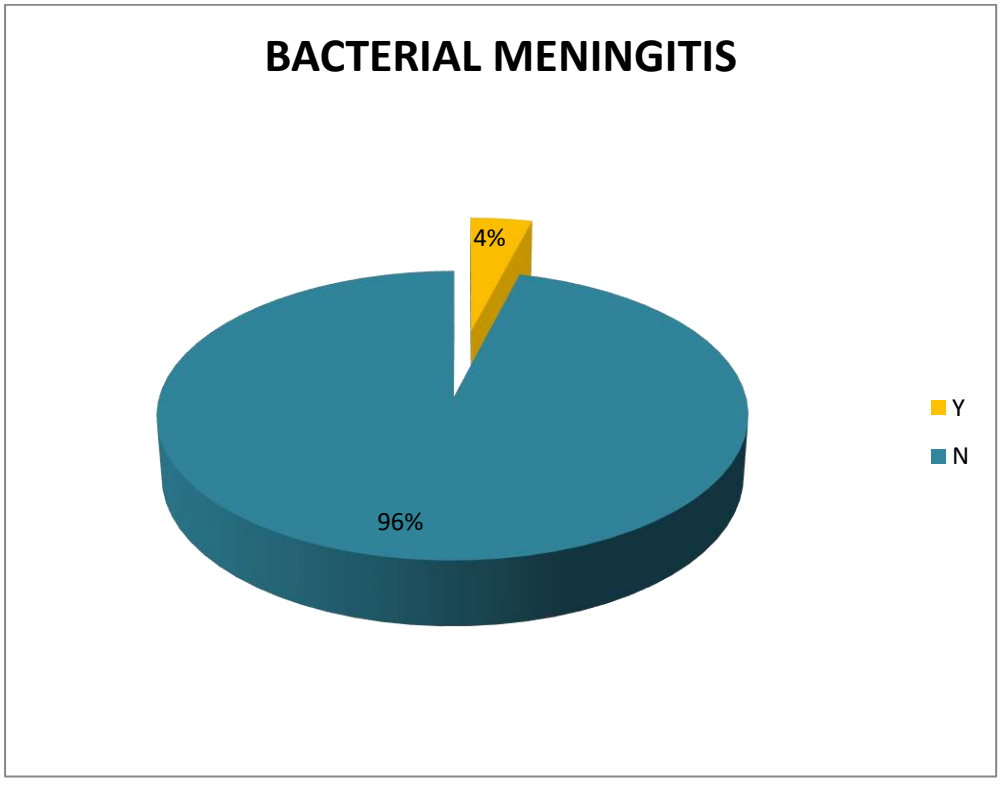
VII) BACTERIAL MENINGITIS

Out of 50 neonates, 2 (4%) had bacterial meningitis and 48 neonates (96%) had no history of bacterial meningitis.(table 26, figure 27)

Table 26: Distribution of Bacterial Meningitis in neonates

Bacterial meningitis	No. of cases	Percentage
No	48	96.0%
Yes	2	4.0%
Total	50	100.0%

Figure 27: Distribution of Bacterial Meningitis in neonates



VIII) PERINATAL ASPHYXIA

(at APGAR of 5 or less at 1min and 6 or less at 5 mins)

Out of the total study population, perinatal asphyxia could be seen in 19 (38%) neonates with APGAR score of 5 or less at 1 min and 14 neonates (28%) had an APGAR of 6 or less at 5 mins.(Table 27, Table 28 , figure 28)

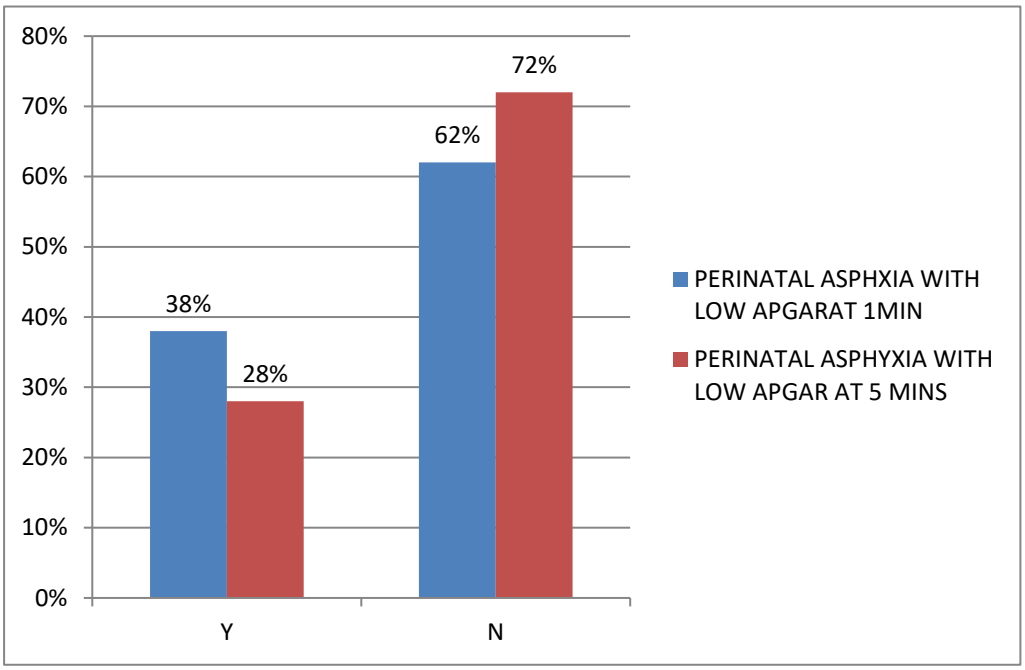
Table 27: Distribution of perinatal asphyxia with low APGAR at 1 min in neonates

Perinatal Asphyxia APGAR At 1 min	No. of cases	Percentage
No	31	62.0%
Yes	19	38.0%
Total	50	100.0%

Table 28: Distribution of perinatal asphyxia with low APGAR at 5 mins in neonates

Perinatal Asphyxia APGAR At 5 min	No. of cases	Percentage
No	36	72.0%
Yes	14	28.0%
Total	50	100.0%

Figure 28: Distribution of perinatal asphyxia with low APGAR at 1 and 5 mins in neonates



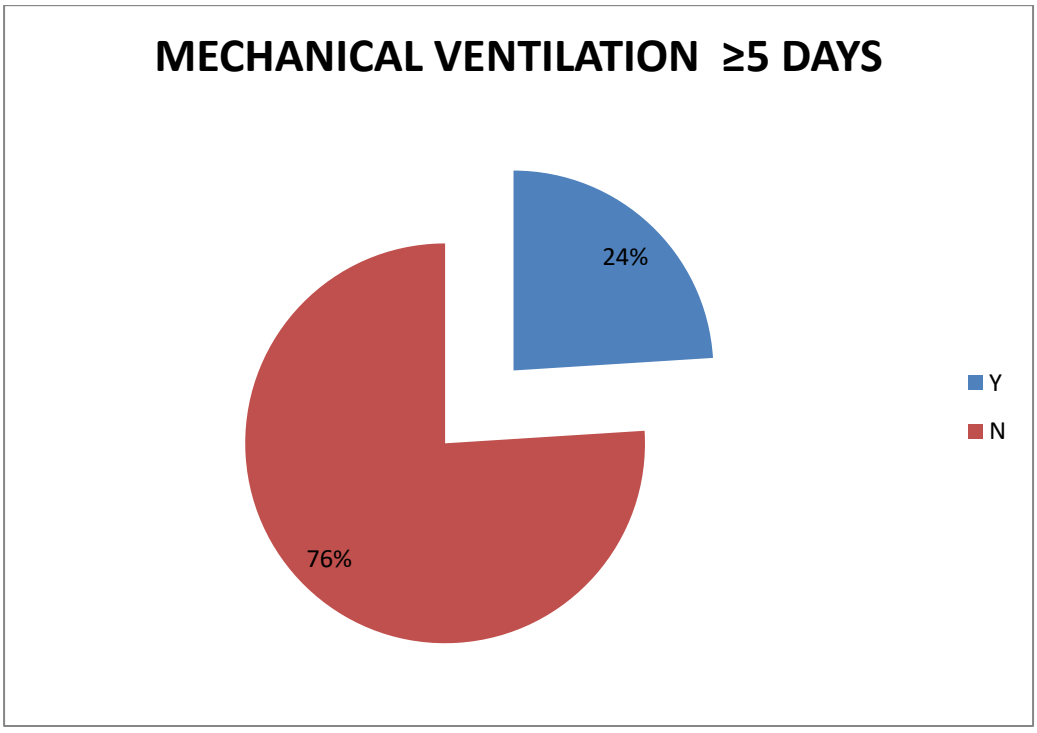
IX) MECHANICAL VENTILATION (≥ 5 days)

12 neonates (24%) out of the total were subjected to ventilatory support for 5 days or more. 38 neonates (76%) were not given ventilatory aid for 5 days or more.(Table 29, Figure 29)

Table 29: Distribution of need for mechanical ventilation ≥5 days in neonates

Mechanical Ventilation ≥ 5days	No. of cases	Percentage
No	38	76.0%
Yes	12	24.0%
Total	50	100.0%

Figure 29: Distribution of need for mechanical ventilation ≥5 days in neonates



X) Stigmata or other findings associated with a syndrome known to include a sensorineural hearing loss.

In our study there was no neonate with stigmata or syndromes associated with SNHL.

6) Examination :

A thorough examination was done for the patients before screening to look for any abnormality. It included general physical examination and local examination.

22 Neonates (44%) had abnormal results and showed pallor/icterus/cyanosis whereas 28 neonates (56%) had normal general physical examination findings.(table 30)

Table 30: General Physical Examination

GENERAL PHYSICAL EXAMINATION	No. of cases	Percentage
Abnormal (pallor/icterus/cyanosis)	22	44.0%
Normal	28	56.0%
Total	50	100.0%

48 neonates (96%) of total had normal tympanic membrane with external ear examination. 2(4%) had B/L ear wax which was cleaned using drops first and then procedure performed.(Table 31)

Table 31: Local examination of Ear

LOCAL EXAMINATION EAR	No. of cases	Percentage
NORMAL	48	96.0%
WAX B/L	2	4.0%
Total	50	100.0%

7) Investigation results :

- CSF gram staining/ culture sensitivity findings:

1 neonate (2%) had enterococcus faecium, 1 neonate (2%) had streptococcus pneumonia in the CSF culture. 9 neonates (18%) showed no growth. In 39 neonates (78%) there was no indication of CSF culture/gram staining. Table 32

Table 32: CSF gram staining/ culture sensitivity results

CSF gram staining/culture sensitivity	No. of cases	Percentage
NA	39	78.0%
no growth	9	18.0%
Streptococcus pneumoniae	1	2.0%
Enterococcus faecium	1	2.0%
Total	50	100.0%

Blood culture: Blood culture was done in all neonates. 1(2%) neonate showed acinetobacter baumanii in the culture, 1 neonate (2%) showed staphylococcus hemolyticus, 48 neonates (96%) showed no growth. Table 33

Table 33: Blood culture results.

BLOOD CULTURE	No. of cases	Percentage
Acinetbacter baumanii	1	2.0%
No growth	48	96.0%
Staphylococcus hemolyticus	1	2.0%
Total	50	100.0%

- **Blood investigations**

The following results were seen on routine blood investigations of the patient.

Serum bilirubin ranged from 0.67-29.6 units was done in 33 neonates, CSF protein done in 8 neonates ranged from 42-156 units, CSF glucose done in 8 neonates ranged from 28-95, haemoglobin done in all neonates ranged from 6.9 to 21.9, total leucocyte count(TLC) ranged from 1.4-28.24 done in all neonates and C reactive protein (CRP) done in all neonates ranged from 0.05-78.4. (TABLE 34)

Table 34: Investigation results

	No of cases	**Minimum value**	**Maximum value**
SERUM BILIRUBIN	33	0.67	29.60
CSF Protein	8	42.00	156.30
CSF glucose	8	28.00	95.00
HEMOGLOBIN	50	6.90	21.90
TLC	50	1.40	28.24
CRP	50	0.05	78.40

8) OAE and BERA testing:

Out of total study population, OAE stage I results as PASS were seen in 22 neonates (44%) whereas OAE stage I results as REFER were seen in 28 neonates (56%). These 28 neonates with Stage I test results as REFER underwent Stage II OAE. Out of 28 neonates(56% of total study population) , 17(34%) neonates gave OAE stage II results as PASS and 11(22%) neonates gave OAE stage II results as REFER. The overall OAE results as PASS were seen in 39 neonates (78%) and overall OAE results as REFER were seen in 11 neonates (22%).

In these 11 neonates, BERA was performed. BERA showed hearing loss in 3(6% of total study population) of these neonates and no hearing loss in other 7 neonates (16% of total population).

(Figure :30- 34, Table: 35-38)

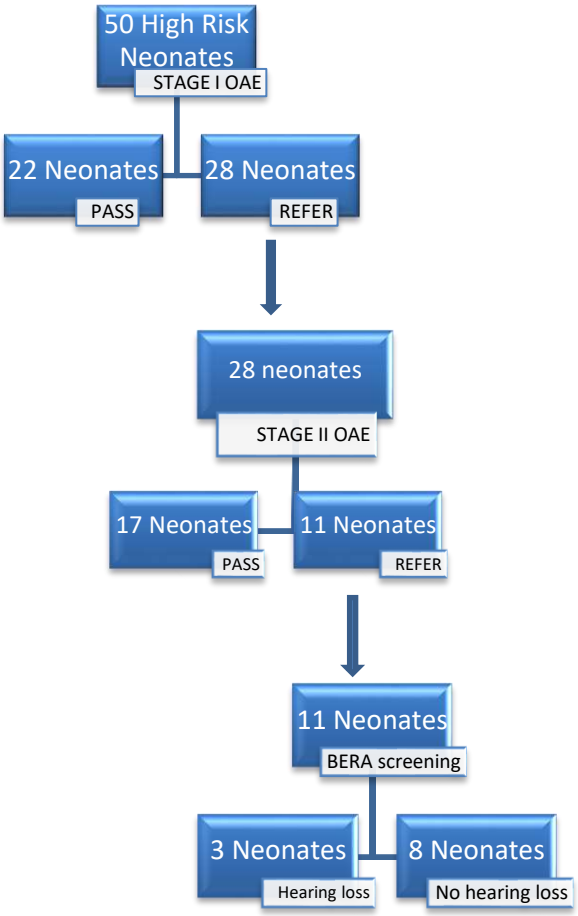


Figure 30: The illustrations depicting the flow of events and outcomes during the neonatal hearing screening.

The test results of OAE Stage I have been documented in the table given below (Table 35 and shown in Figure 31).

Table 35: OAE stage I results

OAE STAGE 1	No. of cases	Percentage
PASS	22	44.0%
REFER	28	56.0%
Total	50	100.0%

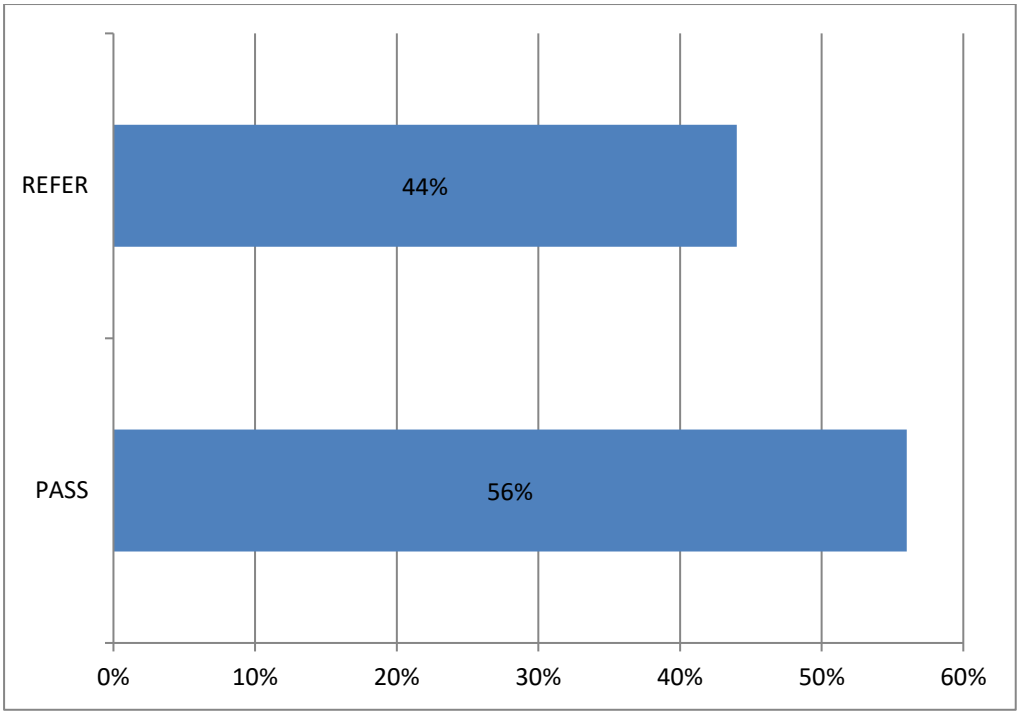


Figure 31: OAE Stage I results

Table 36: OAE stage II results

OAE STAGE II	No. of cases	Percentage
PASS	17	34.0%
REFER	11	22.0%
Total	28	56.0%

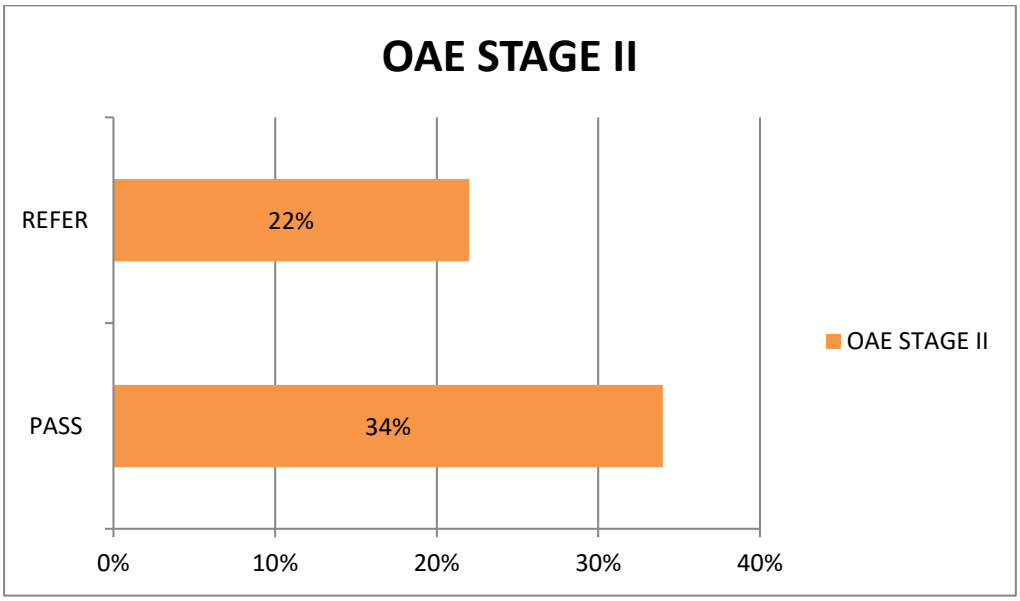


Figure 32: OAE STAGE II results

TABLE 37: OAE results (overall)

OAE	No. of cases	Percentage
PASS	39	78.0%
REFER	11	22.0%
Total	50	100.0%

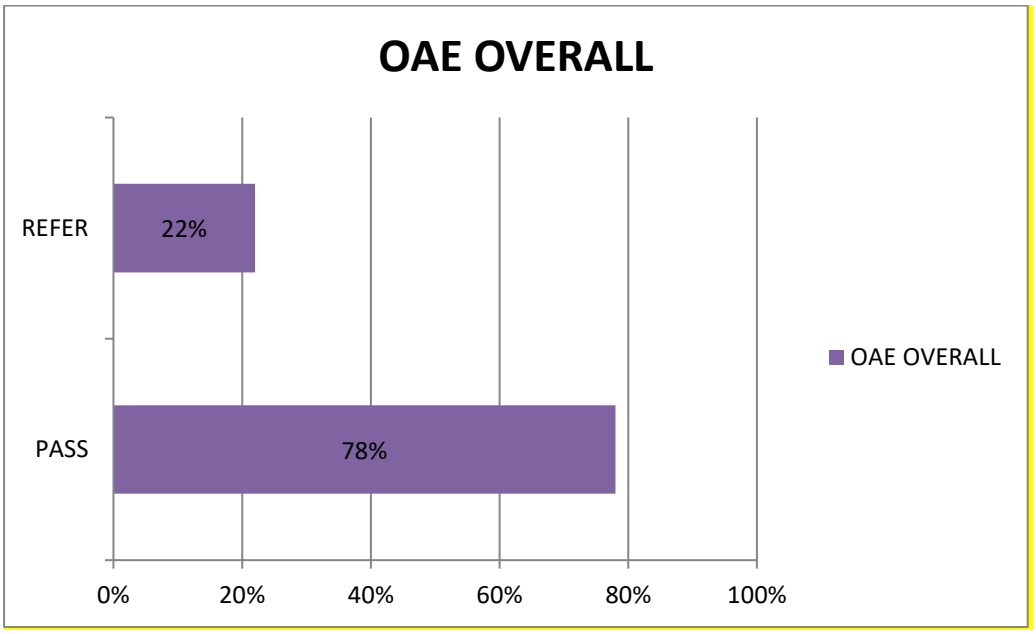
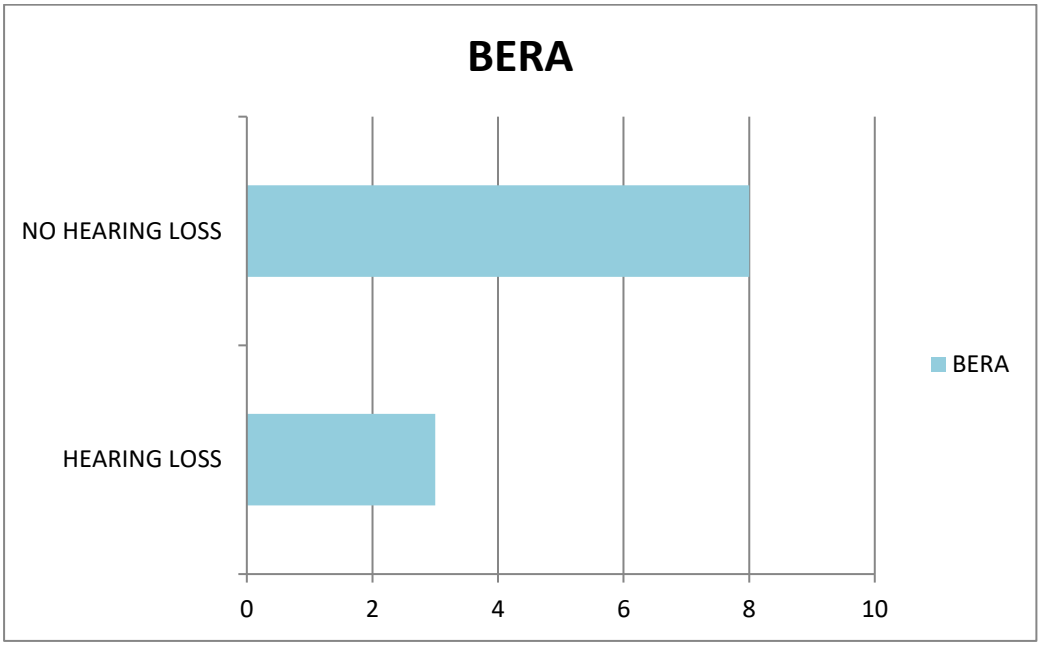


Figure 33: OAE test results (Overall)

Table 38: BERA results

BERA	No. of cases	Percentage
hearing loss	3	6.0%
no hearing loss	8	16.0%
Total	11	22.0%

Figure 34: BERA results



The overall incidence of hearing loss in our study was 6% (3 out 50 neonates). Amongst the neonates with hearing loss, the common high risk factors observed were family history of hearing loss (2 out of 3 neonates), TORCH infections (1 out of 3 neonates), low birth weight in all 3 neonates, hyperbilirubinemia in 1 neonate, use of ototoxic medications in 1 neonate, perinatal asphyxia in 2 neonates and requirement of mechanical ventilation >5 days in 1 neonate. This has been tabulated below:(table 39)

Table 39: Distribution of high risk factors in neonates with hearing loss

High risk factor	**Family history of hearing loss**	**TORCH infections**	**Low birth weight**	**Hyper-bilirubinemia**	**Use of ototoxic medications**	**Perinatal asphyxia**	**Requirement of mechanical ventilation >5 days**
No. of neonates with hearing loss	2	1	3	1	1	2	1

Comparison of outcome with the variables:

A) DEMOGRAPHIC PROFILE

I) AGE OF NEONATES

The age of neonates in our study population ranged from 1-28 days. The mean age of presentation was 8.98 days (S.D.8.95). (Table 40)

Table 40: Age of neonates (range, mean and standard deviation)

	N	Minimum	Maximum	Mean	Std. Deviation
Age (Days)	50	1.00	28.00	8.98	8.95

Overall OAE results of PASS were seen in 39 neonates. Out of these, 21 neonates (53.8% of neonates with PASS result) were of <7 days, 7 (17.9%) were of 8-14days and 11(28.2%) were of >14days of age. Overall OAE results of REFER was seen in 11 neonates. Out of these, 5 neonates (45.5%) were of <7days, 3(27.3%) were 8-14days and 3 (27.3%) were >14days of age.(table 41)

Table 41: Statistical analysis of Age of neonates.

OAE		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
AGE GROUP	< 7 Days	21	53.8%	5	45.5%	26	0.492	0.782
	8-14 Days	7	17.9%	3	27.3%	10		
	> 14 Days	11	28.2%	3	27.3%	14		
Total		39	100.0%	11	100.0%	50		

The statistical analysis was done using chi square and the results were not significant. (Chisquare value: 0.492, p value: 0.782).

II) GENDER

Out of all the neonates with PASS results, 19 neonates (48.7%) were females and 20 neonates (51.3%) were males. And out of all with results as REFER, 4 neonates (36.4%) were females and 7(63.6%) were males. The comparison of gender with study outcome using chisquare was not significant.(chisquare value: 0.527, p value :0.468) (table 42)

Table 42: Statistical Analysis of gender

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Gender	Females	19	48.7%	4	36.4%	23	0.527	0.468
	Males	20	51.3%	7	63.6%	27		
Total		39	100.0%	11	100.0%	50		

III) AGE OF PARENTS

The age of parents in our study population ranged from 21-36 years in mothers and 23-38 years in fathers. The mean maternal age was 25.98 years (S.D.2.65). The mean age of fathers was 27.24 years (S.D. 2.54) (Table 43)

Table 43: Statistical analysis of age of parents (range, mean and standard deviation)

In years	N	Minimum	Maximum	Mean	Std. Deviation
Mother Age	50	21.00	36.00	25.98	2.65
Father Age	50	23.00	38.00	27.24	2.54

IV) OCCUPATION OF PARENTS

Out of all mothers with OAE results as PASS, 1(2.6%) was baker, 1(2.6%) was house helper, 1(2.6%) was student in college, 1(2.6%) was worker, 3(7.7%) were doctors, 24(61.5%) were housewives, 6(15.4%) were teachers, 2(5.1%) were doing service. And mothers of neonates with OAE results as REFER 10(90.9%) were housewives and 1(9.1%) was teacher.

On application of chisqaure for statistical analysis the results were not significant (chi square value: 3.87, p value: 0.795) Table 44

Table 44: Statistical analysis of age of mother with study outcomes.

		PASS		**REFER**		**Total**	**Chi-square value**	**p-value**
		No. of cases	**%age**	**No. of cases**	**%age**			
Mother occupation	BAKER	1	2.6%	0	0.0%	1	3.87	0.795
	DOCTOR	3	7.7%	0	0.0%	3		
	HOUSEHELPER	1	2.6%	0	0.0%	1		
	HOUSEWIFE	24	61.5%	10	90.9%	34		
	SERVICE	2	5.1%	0	0.0%	2		
	STUDENT IN COLLEGE	1	2.6%	0	0.0%	1		
	TEACHER	6	15.4%	1	9.1%	7		
	WORKER	1	2.6%	0	0.0%	1		
Total		39	100.0%	11	100.0%	50		

Table 45: Statistical analysis of father's occupation with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Father occupation	SERVICE	5	12.8%	0	0.0%	5	21.809	0.083
	TEACHER	1	2.6%	0	0.0%	1		
	BUSINESSMAN	13	33.3%	3	27.3%	16		
	CLERK	0	0.0%	1	9.1%	1		
	DOCTOR	2	5.1%	0	0.0%	2		
	ENGINEER	1	2.6%	0	0.0%	1		
	EVENT ORGANISER	1	2.6%	0	0.0%	1		
	FACTORY WORKER	2	5.1%	0	0.0%	2		
	FARMER	0	0.0%	2	18.2%	2		
	JOB	2	5.1%	0	0.0%	2		
	LABOURER	1	2.6%	0	0.0%	1		
	MARKETING	1	2.6%	0	0.0%	1		
	SERVICE	4	10.3%	0	0.0%	4		
	SHOPKEEPER	6	15.4%	4	36.4%	10		
	TAILOR	0	0.0%	1	9.1%	1		
Total		39	100.0%	11	100.0%	50		

Out of all the fathers with OAE results as PASS , 5(12.8%) were doing service, 1(2.6%) was teacher, 13(33.3%) were businessmen, 2(5.1%) were factory worker, 2(5.1%) were doctors,1(2.6%) was engineer, 1(2.6%) was event organiser, 1(2.6%) was labourer, 1(2.6%) was in a marketing firm, , 2(5.1%) were doing job,4(10.3%) were in service and 6 (15.4%) were shopkeepers and fathers of neonates with OAE results as REFER 1(9.%) was tailor,4(36.4%) were shopkeepers,2 (18.2%) were farmers,3(27.3%) were businessmen and 1(9.1%) was a clerk.

On application of chisqaure for statistical analysis the results were not significant (chi square value: 21.809, p value: 0.083) table 45

V) AREA WISE DISTRIBUTION

Out of the 39 neonates with OAE results as PASS, 10(25.6%) were from rural area and 29(74.4%) were from urban area. And from the 11 candidates in whom OAE results were REFER, 5 (45.5%) were from rural area and 6 (54.5%) were from an urban area. The chi square value calculated was 1.604 with a p value of 0.205 (not significant) table 46

Table 46: Statistical analysis of community distribution with the outcomes of screening.

		PASS		**REFER**		**Total**	**Chi-square value**	**p-value**
		No. of cases	**%age**	**No. of cases**	**%age**			
urban/rural	RURAL	10	25.6%	5	45.5%	15	1.604	0.205
	URBAN	29	74.4%	6	54.5%	35		
Total		39	100.0%	11	100.0%	50		

B) CLINICAL PROFILE

I) BIRTH ORDER

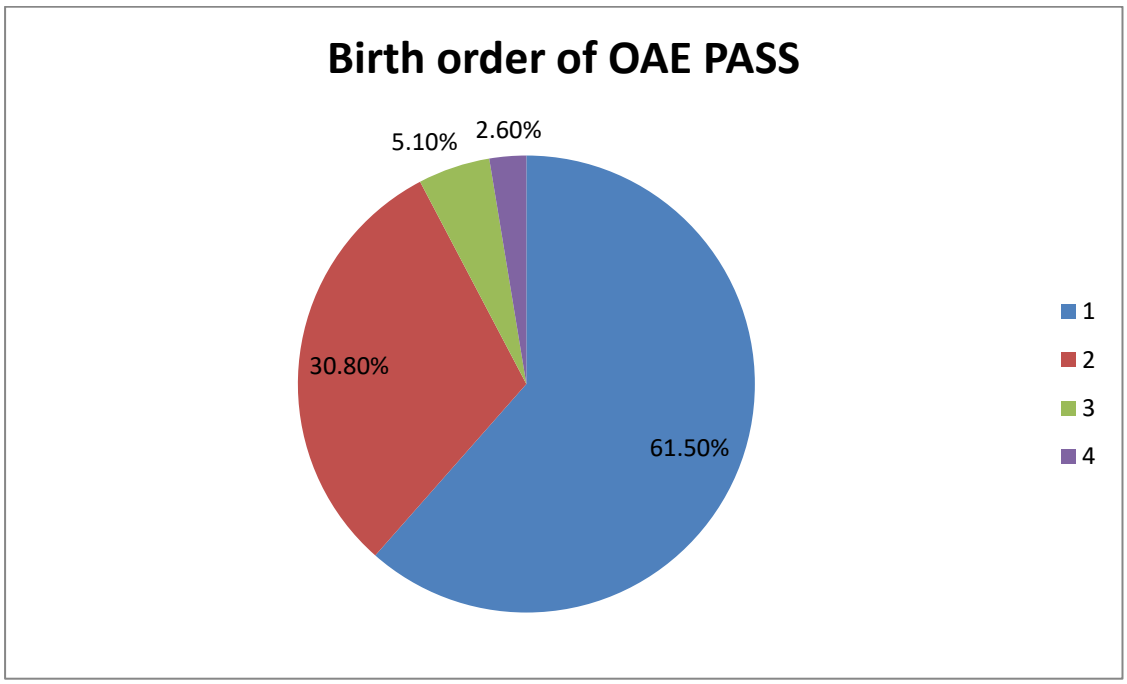
In neonates with OAE results as PASS, a birth order of 1 was seen in 24(61.5%) neonates, birth order 2 in 12(30.8%), birth order 3 in 2 (5.1%) and a birth order of 4 in 1(2.6%) whereas in neonates in whom the results were REFER, the distribution of neonates was 4 (36.4%), 6 (54.5%), 1 (9.1%) of birth order 1, 2 and 3 respectively.

Statistically it was not significant. (Chisquare value: 2.825 p value: 0.419) (Table 47, Figure 35)

Table 47: Statistical analysis of birth order

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Birth Order	1	24	61.5%	4	36.4%	28	2.825	0.419
	2	12	30.8%	6	54.5%	18		
	3	2	5.1%	1	9.1%	3		
	4	1	2.6%	0	0.0%	1		
Total		39	100.0%	11	100.0%	50		

Figure 35: Birth order in OAE PASS.



II) APGAR

Out of 47 neonates with known APGAR score, the range at 1min was 3-9 with a mean value of 6.36 and S.D. 1.7 and at 5 mins it was from 5-10 with mean of 8.02 and S.D. 1.5

Table 48: Mean and standard deviation of APGAR scores.

	No of cases with known APGAR score	**Minimum**	**Maximum**	**Mean**	**Std. Deviation**
APGAR score At 1 min	47	3.00	9.00	6.36	1.70
APGAR score At 5 mins	47	5.00	10.00	8.02	1.50

In 47 neonates the APGAR score was known. Out of these, a score of 3 was minimum and 9 maximum at 1 min and a score of 5 was minimum and 10 maximum at 5 mins. The mean value calculated AT 1min was 6.36 (S.D. 1.70) and at 5 mins 8.02 (S.D. 1.50). (table 48)

Neonates at with low APGAR score at 1min with OAE results as PASS had the mentioned distribution. An APGAR score of 3 was seen in 4(10.3%),5 in 4(10.3%),6 in 3 (7.7%),7 in 12(30.8%),8 in 13(33.3%) and 9 in 1(2.6%)neonates and at 5mins , APGAR score of 5 was seen in 3(7.7%), 6 in 1(2.6%), 7 in 3(7.7%), 8 in 12(30.8%), 9 in 11(28.2%) and 10 in 7(17.9%). The statistical analysis was significant (chisquare value: 14.751 p value 0.022 at 1min) and chisquare value :16.509 p value 0.006). Table 49 (a,b)

Table 49: APGAR score at 1min and 5 mins.

Table 49 a: Statistical analysis of APGAR score at 1min.

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
APGAR score At 1 min	3	4	10.3%	1	9.1%	5	14.751	0.022
	4	0	0.0%	2	18.2%	2		
	5	4	10.3%	4	36.4%	8		
	6	3	7.7%	1	9.1%	4		
	7	12	30.8%	1	9.1%	13		
	8	13	33.3%	1	9.1%	14		
	9	1	2.6%	0	0.0%	1		
Total		37	94.9%	10	90.9%	47		

Table 49 b: Statistical analysis of APGAR score at 5 mins.

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
APGAR score At 5 mins	5	3	7.7%	1	9.1%	4	16.509	0.006
	6	1	2.6%	4	36.4%	5		
	7	3	7.7%	2	18.2%	5		
	8	12	30.8%	0	0.0%	12		
	9	11	28.2%	3	27.3%	14		
	10	7	17.9%	0	0.0%	7		
Total		37	94.9%	10	90.9%	47		

I) ANTENATAL HISTORY

- In neonates with test results as PASS, 5(12.8%) had a positive antenatal history of hypertension and amongst the neonates with test results as REFER, 1 (9.1%) had a positive antenatal history of hypertension. The statistical analysis done using chisquare was not significant.(chi square value : 0.421 , p value 0.810). table 50
- In neonates with test results as PASS, 3(7.7%) had a positive antenatal history of pre eclampsia and amongst the neonates with test results as REFER, 3 (27.3%) had a positive antenatal history of pre eclampsia. The statistical analysis done using chisquare was not significant.(chi square value 3.312 :, p value 0.191). Table 51
- In neonates with test results as PASS, 2(5.1%) had a positive antenatal history of diabetes and amongst the neonates with test results as REFER,1(9.1%) had a positive antenatal history of diabetes. The statistical analysis done using chisquare was not significant. (chi square value:0.508, p value :0.776).Table 52
- In neonates with test results as PASS,5(12.8%)had a positive antenatal history of thyroid and amongst the neonates with test results as REFER, all the 11 neonates had a negative antenatal history of thyroid. The statistical analysis done using chisquare was not significant.(chi square value : 1.923, p value: 0.382). table 53
- In neonates with test results as PASS,1(2.6%) had a positive antenatal history of epilepsy and amongst the neonates with test results as REFER, all the 11 neonates had a negative antenatal history of epilepsy. The statistical analysis done using chisquare was not significant.(chi square value : 0.588 , p value:0.745) table 54
- In neonates with test results as PASS, 3 (7.7%) had a positive antenatal history of sensorineural hearing loss and amongst the neonates with test results as REFER, 1 (9.1%) had a positive antenatal history of sensorineural hearing loss. The statistical analysis done using chisquare was not significant.(chi square value 0.304, p value: 0.859).Table 55

Table 50: Statistical analysis of history of hypertension with the study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
H/O Hypertension	No	33	84.6%	10	90.9%	43	0.421	0.810
	Not Known	1	2.6%	0	0.0%	1		
	Yes	5	12.8%	1	9.1%	6		
Total		39	100.0%	11	100.0%	50		

Table 51: Statistical analysis of history of pre eclampsia with the study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
H/o preeclampsia	No	35	89.7%	8	72.7%	43	3.312	0.191
	Not Known	1	2.6%	0	0.0%	1		
	Yes	3	7.7%	3	27.3%	6		
Total		39	100.0%	11	100.0%	50		

Table 52: Statistical analysis of history of diabetes with the study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
h/o Diabetes	No	36	92.3%	10	90.9%	46	0.508	0.776
	Not Known	1	2.6%	0	0.0%	1		
	Yes	2	5.1%	1	9.1%	3		
Total		39	100.0%	11	100.0%	50		

Table 53: Statistical analysis of history of thyroid disorders with the study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
H/o thyroid disorders	No	33	84.6%	11	100.0%	44	1.923	0.382
	Not Known	1	2.6%	0	0.0%	1		
	Yes (OVERT)	5	12.8%	0	0.0%	5		
Total		39	100.0%	11	100.0%	50		

Table 54: Statistical analysis of history of epilepsy with the study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
H/o Epilepsy	No	37	94.9%	11	100.0%	48	0.588	0.745
	Not Known	1	2.6%	0	0.0%	1		
	Yes	1	2.6%	0	0.0%	1		
Total		39	100.0%	11	100.0%	50		

Table 55: Statistical analysis of history of sensorineural hearing loss with the study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
H/o Sensorineural hearing loss	No	35	89.7%	10	90.9%	45	0.304	0.859
	Not Known	1	2.6%	0	0.0%	1		
	Yes	3	7.7%	1	9.1%	4		
Total		39	100.0%	11	100.0%	50		

II) BIRTH HISTORY

Place of delivery when compared with the outcomes of screening test using chisquare, the results were not significant (p value 0.592, chisquare value 0.288) table 56

Table 56: Statistical analysis of place of delivery

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Place of Delivery	HOSPITAL	38	97.4%	11	100%	49	0.288	0.592
	HOME	1	2.6%	0	0.0%	1		
Total		39	100.0%	11	100.0%	50		

Table 57: Statistical analysis of type of delivery.

Type of delivery is also not significant for the outcomes of the disease.(chi square value:0.228 p value:0.633). Table 57

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Type of Delivery	LSCS	31	79.5%	8	72.7%	39	0.228	0.633
	NVD	8	20.5%	3	27.3%	11		
Total		39	100.0%	11	100.0%	50		

V) HIGH RISK FACTORS and HEARING LOSS

A) Family history of SNHL

Out of total neonates with OAE results as PASS, 11 (28.2%) had a positive family history of sensorineural hearing loss whereas 3 neonates (27.3%) amongst the OAE results as REFER had a positive family history of SNHL.

The statistical analysis done using the chisquare method (value 0.004) with p value (0.951) was not significant.(TABLE 58)

Table 58: Statistical analysis of family history of sensorineural hearing loss with study outcomes.

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Family history of SNHL	NO	28	71.8%	8	72.7%	36	0.004	0.951
	YES	11	28.2%	3	27.3%	14		
Total		39	100.0%	11	100.0%	50		

B) TORCH infections

Out of the 39 neonates, 6 (15.4%) neonates with OAE test results as PASS had h/o TORCH infection whereas 33(84.6%) had no such history. Out of 11 neonates with test results as REFER, 2(18.2%) had a positive history of TORCH infections and 9 (81.8%) had no such history.

The statistical analysis done using chi square method was not significant.(chisquare value :0.05and p value of 0.823). Table 59

Table 59: Statistical analysis of history of TORCH infections with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
TORCH Infections)	NO	33	84.6%	9	81.8%	42	0.05	0.823
	YES	6	15.4%	2	18.2%	8		
Total		39	100.0%	11	100.0%	50		

C) CRANIOFACIAL ANAMOLIES:

Out of the 39 neonates, 1 (2.6%) neonates with OAE test results as PASS had h/o Craniofacial anomaly whereas 38(97.4%) had no such history. Out of 11 neonates with test results as REFER, no such patient was found.

Statistical analysis done using chisquare method was not significant. (chisqaure value: 0.288 and p value: 0.592) Table 60

Table 60: Statistical analysis of history of Craniofacial Anamolies with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Craniofacial Anomalies	NO	38	97.4%	11	100.0%	49	0.288	0.592
	YES	1	2.6%	0	0.0%	1		
Total		39	100.0%	11	100.0%	50		

D) LOW BIRTH WEIGHT

Out of the 39 neonates,29(74.4%) neonates with OAE test results as PASS had h/o low birth weight whereas 10(25.6%) had no such history. Out of 11 neonates with test results as REFER, 10(90.9%) had a positive history of low birth weight and 1 (9.1%) had no such history.

The statistical analysis done using chi square method was not significant.(Chisquare value :1.370 p value : 0.242) Table 61

Table 61: Statistical Analysis history of low birth weight with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
low birth weight	NO	10	25.6%	1	9.1%	11	1.370	0.242
	YES	29	74.4%	10	90.9%	39		
Total		39	100.0%	11	100.0%	50		

E) HYPERBILIRUBINEMIA (with serum levels requiring exchange transfusions)

Out of the 39 neonates, 2(5.1%) neonates with OAE test results as PASS had h/o hyperbilirubinemia whereas 37(94.9%) had no such history. Out of 11 neonates with test results as REFER, 3(27.3%) had a positive history of hyperbilirubinemia and 8 (72.7%) had no such history.

The statistical analysis done using chi square method was not significant.(Chisquare value of 4.675 and p value of 0.064)Table 62

Table 62: Statistical analysis of history of hyperbilirubinemia with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
hyperbilirubinemia	NO	37	94.9%	8	72.7%	45	4.675	0.064
	YES	2	5.1%	3	27.3%	5		
Total		39	100.0%	11	100.0%	50		

F) OTOTOXIC MEDICATIONS

Out of the 39 neonates,2(5.1%) neonates with OAE test results as PASS had h/o use of ototoxic medications whereas 37(94.9%) had no such history. Out of 11 neonates with test results as REFER, 1(9.1%) had a positive history of use of ototoxic medications and 10 (90.9%) had no such history.

The statistical analysis done using chi square method was not significant.(chisquare value 0.239, p value 0.625) table 63

Table 63: Statistical analysis of history of use of ototoxic medications with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Ototoxic medications	No	37	94.9%	10	90.9%	47	0.239	0.625
	Yes	2	5.1%	1	9.1%	3		
Total		39	100.0%	11	100.0%	50		

G) BACTERIAL MENINGITIS

Out of the 39 neonates, 2(5.1%) neonates with OAE test results as PASS had h/o bacterial meningitis whereas 37(94.9%) had no such history. Out of 11 neonates with test results as REFER, no such positive history was found.

The statistical analysis done using chi square method was not significant. (chisquare value 0.588, p value 0.443) Table 64

Table 64: Statistical analysis history of bacterial meningitis with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
bacterial meningitis	No	37	94.9%	11	100.0%	48	0.588	0.443
	Yes	2	5.1%	0	0.0%	2		
Total		39	100.0%	11	100.0%	50		

H) PERINATAL ASPHYXIA (at APGAR of 5 or less at 1min and 6 or less at 5 mins)

Out of the 39 neonates, 11(28.2%) with OAE test results as PASS had h/o perinatal asphyxia at a low APGAR at 1 min whereas 28(71.8%) had no such history. Out of 11 neonates with test results as REFER, 8(72.7%) had a h/o perinatal asphyxia at a low APGAR at 1 min
And 3(27.3%) had no such positive history

The statistical analysis done using chi square method was statistically significant.(chisquare value 7.219, p value 0.007) Table 65(i)

Out of the 39 neonates, 7(17.9%) with OAE test results as PASS had h/o perinatal asphyxia at a low APGAR at 5 mins whereas 32(82.1%) had no such history. Out of 11 neonates with test results as REFER, 7 (63.6%) had a h/o perinatal asphyxia at a low APGAR at 5mins and 4 (36.4%) had no such positive history

The statistical analysis done using chi square method was statistically significant.(chisquare value 8.884, p value 0.003) Table 65(ii)

Table 65(i, ii): Statistical Analysis of history of perinatal asphyxia at a low APGAR score with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
i) Perinatal Asphyxia at low APGAR At 1 min	No	28	71.8%	3	27.3%	31	7.219	0.007
	Yes	11	28.2%	8	72.7%	19		
Total		39	100.0%	11	100.0%	50		
ii) Perinatal Asphyxia at low APGAR At 5 mins	No	32	82.1%	4	36.4%	36	8.884	0.003
	Yes	7	17.9%	7	63.6%	14		
Total		39	100.0%	11	100.0%	50		

I) MECHANICAL VENTILATION (for ≥5 days)

Out of the 39 neonates, 7(17.9%) with OAE test results as PASS had h/o mechanical ventilation ≥5days whereas 32(82.1%) had no such history. Out of 11 neonates with test results as REFER, 5(45.5%) had a h/o mechanical ventilation ≥5days and 6 (54.5%) had no such positive history.

The statistical analysis done using chi square method was not significant.(chisquare value 3.559, p value 0.105) Table 66

Table 66: Statistical analysis of need for mechanical ventilation ≥ 5 days with study outcomes

		PASS		REFER		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Mechanical Ventilation ≥5days	No	32	82.1%	6	54.5%	38	3.559	0.105
	Yes	7	17.9%	5	45.5%	12		
Total		39	100.0%	11	100.0%	50		

DISCUSSION

The perception of hearing and development of language are essential for the normal functioning of human being. The process of development begins early in the fetal stage and continues upto early childhood. Hence it becomes important to screen the newborns for hearing impairment and commence the early intervention wherever possible. We conducted a prospective observational study which included 50 high risk neonates as per the inclusion criteria. These neonates underwent a two staged OAE screening followed by BERA (if indicated).

Demographic profile:

In our study, maximum patients were <7 days of age [26 neonates (52%)]. There were 10(20%) neonates of 8-14 days of age and 14 neonates (28%) >14 days of age. The mean age was 8.98 days.

In a study conducted by Kamath R. out of the 425 neonates, 250 (58.82%) were 3-7 days, 78 (18.35%) were 8-14days mean age was 9.42 days and 97(22.82%) were >15 days of age.[79]

In our study, 23 neonates (46%) were females and 27(54%) were males.

In a study conducted by Raghuwanshi SK et al the ratio of males was more than females [659 neonates (52.72%) were males and 591 neonates (47.28 %) were females][4] which is similar to our study.

In another study by Kaveh M et al, 99 (56.9%) were males and 75(43.1%) were females.[14]

There were 253 (50.5%) males and 212 (45.5%) females in a study by Khaimook W et al.[77]

The maternal age and age of fathers in our study ranged from 21-36 years and 23-38 years respectively.

We observed a spectrum of occupations in fathers. 16(32%) fathers were businessmen, hence being the most common occupation.

The mothers also had varied occupations but most of them were housewives (34 mothers, 68%)

15 neonates (30%) were from rural area and 35(70%) from urban area.

Clinical profile:

We observed that the most common birth order in our study group was 1. It was seen in 28(56%) neonates. Birth order of 2 was seen 18 (36%), 3 in 3 (6%) and 4 in 1 (2%) neonate.

The APGAR score for the neonates was documented at 1 min and 5 mins of birth.

At 1 min, the most commonly observed APGAR score was 8. It was seen in 14(28%) neonates followed by an APGAR score of 7 in 13(26%), 5 in 8(16%), 3 in 5(10%), 6 in 4(8%), 9 in 3(6%) and 4 in 2(4%). It was unknown in 3 (6%) neonates.

At 5 mins, the most common APGAR score observed was 9, seen in 14(28%) neonates followed by an APGAR score of 8 in 12(24%), 10 in 7(14%), 7 in 5(10%), 6 in 5(10%), and 5 in 4(8%). It was unknown in 3 (6%) neonates.

In a study by Kaveh M et al, the most common APGAR score observed at 1min was 8, seen in 13(33.3%) neonates followed by 7 in 12 (30.8%), 5 in 4(10.3%), 3 in 4(10.3%), 6 in 3(7.7%), and 9 in 1(2.6%)neonates [14]

Whereas at 5 mins, an APGAR score of 8 in 12(30.8%) was most common followed by 9 in 11(28.2%), 10 in 7(17.9%), 7 in 3 (7.7%), 5 in 3(7.7%) and 6 in 1(2.6%) neonates.[14]

In another study by. Zhu Y et al, amongst 1460 neonates, an APGAR score at 1min was <4 in 3(0.2%) neonates, 4-6 in 24(1.6) and 7-10 in 1433 (98.2%) neonates.[80]

Similarly at 5 mins an APGAR score of < 4-6 in 1(0.1%) and 7-10 in 1459 (99.1%) neonates[80]

The various parameters observed while taking the antenatal history of mothers were- history of hypertension, preeclampsia, diabetes, thyroid disorders, epilepsy, drug intake and sensorineural hearing loss.

History of hypertension was positive in 6 mothers (12%), preeclampsia in 6(12%), diabetes in 3(6%), thyroid disorders in 5(10%), epilepsy in 1(2%), history of sensorineural hearing loss in 4(8%) and history of drug intake in 10(20%) mothers.

39 neonates (78%) were born via lower segment caesarean section and 11 (22%) by normal vaginal delivery.

In a study by, Zhu Y et al Among the 1460 infants, 1037(71%) infants were born by vaginal delivery and 423 (29%) by caesarean section.[80]

In a study by De Kock T et al, out of 7451 neonates screened, 5414 (72.7%) were born by normal vaginal delivery.[81]

In a study by A. Suppiej et al, out of 206 children, 51 were born by vaginal delivery and 155 via caesarean section.[82]

In our study, most of the neonates (49 neonates, 98%) were born in hospital except 1 (2%) who was born at home.

High risk factors:

A plethora of high risk factors were included. These were family history of sensorineural hearing loss, TORCH infections, low birth weight (<2500gms), craniofacial anomalies, hyperbilirubinemia at serum levels requiring exchange transfusion, use of ototoxic medications, bacterial meningitis, perinatal asphyxia at a low APGAR

score, need for mechanical ventilation ≥5days and syndromes or stigmata associated with SNHL.

In our study low birth weight was observed to be the most common high risk factor. It was seen in 39 neonates (78%) followed by perinatal asphyxia at a low APGAR [which was seen in 14(28%) neonates at 1 min and 19 (38) neonates at 5 mins].

Other risk factors seen were the presence of family history of SNHL in 14 (28%), need for mechanical ventilation for >5 days in 12 neonates (24%), TORCH infections in 8 neonates (16%), hyperbilirubinemia (with serum levels requiring exchange transfusions) in 5(10%), craniofacial anomaly in 1 (2%), history of use of ototoxic medication in 3(6%), bacterial meningitis in 2 (4%) and craniofacial anomaly in 1 (2%).

In a study by Anand S et al 254 out of 1052 neonates were known to have risk factors. Amongst the high risk factors, history of ototoxic drugs was the most common high risk factor. It was seen in 56 neonates (22%) followed by neonatal hyperbilirubinemia requiring exchange transfusion in 54(21%), asphyxia in 47(18.5%), low birth weight <1.5 kg in 43 neonates (16.9%), meningitis in 32(12.59%), requirement of ventilation in 32 (12.59%) and family history of hearing impairment in 23 (9%) neonates.[70]

Another study by Khaimook W et al 465 neonates were screened. The most common high risk factor observed was prematurity. It was seen in 226 (48.6%) neonates followed by use of ototoxic medications in 159 (34.2%), prolonged intubation in 61 (13.1%), low birth weight in 54(11.6%), craniofacial anomaly in 11 (2.36%), history of in utero infections in 6 (1.29%), hyperbilirubinemia in 6 (1.29%) and syndromes associated with hearing loss in 6(1.29%) neonates.[77]

In a study by Di Stadio A et al, out of 153 neonates screened, 12 neonates had sensorineural hearing loss. The history of SNHL, prematurity (<28 weeks) and low weight at birth were common findings in all subjects, followed by hospitalization in NICU for more than 5 days (in 10 neonates, 83.3%), the use of ototoxic antibiotics in 10 neonates (83.3%), caesarean childbirth in 8 neonates (66.6%), mechanical ventilation over 5 days in 7 neonates (58.3%), retinopathy of prematurity (ROP) in 6 (50%) neonates and surgery in 6 neonates (50%). Less-common findings were twin pregnancy (4, 33.3%) and maternal disease during the pregnancy (4, 33.3%) followed by sepsis (3, 25%), maternal infections (2, 16.7%), chromosomal disorders (2, 16.7%), craniofacial malformations (2, 16.7%) and maternal use of medicines during pregnancy (2, 16.7%).[78]

Outcomes of OAE and BERA :

In our study while performing the OAE screening, we observed that 22(44%) neonates passed the Stage I OAE. The 28 (56%) neonates who failed stage I OAE test, were called for follow up for the stage II OAE screening. In this stage, 17(43%) neonates passed. So the neonates with overall OAE results as PASS were 39(78%) and as REFER were 11(22%). These 11(22%) neonates underwent BERA test. BERA test showed abnormal results in 3 neonates (6% of total) and normal hearing in 8 neonates.

In a study by Paul et al, 10165 newborns were screened. Out of these 2031 neonates were of high risk category and rest 8134 neonates were of non high risk category. Out the high risk neonates, 234 (11.53%) failed the stage I screening using OAE. Out of these, 78(3.84% of high risk neonates) failed the second screen, 140(6.89% of high risk neonates) passed and 16(0.78%) were dropouts. These 78 (3.84% of high risk neonates) neonates who failed the second screening underwent BERA test. 21(1.03%) neonates gave abnormal results on BERA and 57(2.8%) were normal. Hence hearing loss was seen in 21(1.03%) neonates.[58]

In a study by Anand S et al, 1052 babies (562 males and 490 females) were screened, out of which 254 babies had risk factors for hearing impairment. Out of these, 20 babies (7.8%) showed hearing impairment in contrast with our study where overall hearing loss was seen in 6%.[70]

In another study by Abdullah A et al, out of all the deliveries done, 3762 infants were enrolled in the study and subjected to a three staged OAE screening followed by BERA. 620 (16.48% of total) infants failed the first screening test. They were called for second screening. Out of whom only 506 (81.6 % of infants who failed stage I) came for a second stage screening test. 446 infants passed the second screening and 60 failed. These 60 infants were called for the third stage screening at three months of age. Only 39 (65% of infants with failed stage II screening) patients turned up. Of these, 10 (25.64%) infants passed the OAE test and 29(74.3%) failed. However, when these 29 infants underwent BERA, 13(44.8%) had normal BERA and 16(55.17%) had abnormal BERA.[51]

In a study by Raghuwanshi SK et al, 1250 newborns (659 boys and 591 girls) were assessed. In the first phase, out of the 1250 newborns screened using OAE, 1185 (94.8%) had normal and 65 (5.2%) had abnormal results in OAE screening. Repeat OAE assessment was done after 1 month period and 51(78.4% of neonates with abnormal stage I results) newborns passed the second stage OAE, 10(15.38%) had abnormal results and 4(6.15% of neonates with abnormal OAE stage I) patients did not return for follow up visit. Third visit was conducted after 3 months from birth and out of 10 patients with OAE stage II results as REFER, 6(60%) had normal OAE result and 4(40%) neonates had abnormal results. These 4 newborns were screened with BERA. Out of these 4 neonates with OAE stage III results as REFER, 3 (75%) had abnormal BERA results and 1(25%) had normal BERA result. The overall incidence of hearing loss in this study was 0.24% (3/1250 neonates).[4]

In another study by Khaimook W et al, 465 neonates were included. Out of these 416(89%) passed the stage I screening with portable OAE. The 49(11%) neonates who failed the OAE stage I , were taken for screening using conventional OAE. 40(8.6%) neonates passed, 6(1.3%) failed and 3(0.1%) were lost to follow up. These 6 neonates when screened using BERA, showed hearing loss in only 4(0.86%) neonates.[77]

In a study by Kamath R , out of 425 neonates screened, 405(95.29%) passed the first OAE screening. The results were refer in 20(4.7%) neonates, which were subjected to second stage OAE screen. Out of these 20 neonates, 15(75%) neonates passed the stage II and 5(25%) failed. These 5 neonates underwent BERA test. Out of these, 4(80%) neonates showed hearing loss on BERA screen and 1(20%) was lost to follow up. The overall incidence of hearing loss was 0.94% (4/425). [79]

In a study by Di Stadio A et al, out of 153 neonates, 113 neonates (73.9%) had normal hearing results with TEOAE and A-ABR, 15 (9.8%) neonates had abnormal results with TEOAE and a normal result with A-ABR. 25 (16.3%) neonates had abnormal A-ABR response and were addressed to clinical ABR evaluation. The results of clinical ABR showed that 12/25 (48%) children had SNHL while 11/25 (44%) were false positives and had a normal hearing.[78]

Similar study was conducted by Rughani Sudhir et al, in which out of the 306 newborns screened, 100 high risk newborns (22%) showed hearing impairment.[71]

In another study by Bhatt J et al, 500 newborns that were born in hospital were followed up for next 6 months by applying OAE at 0 months, 3 months and BERA at 3 months and 6 months. Out of 500 neonates screened, 100(20%) neonates were at risk and by the end of study, 5(1%) neonates were found to have hearing loss.[72]

In another study by Sachdeva K et al, a total of 2254 subjects were screened using the two staged OAE followed by BERA. Out of total neonates 127(5.6%) subjects had risk factors, 2127(94.3%) were not at risk. 20 (0.88%) high risk subjects were diagnosed to have hearing loss after BERA test.[83]

In a study by Fredy A. Escobar-Ipuz et al, a total of 9390 OAEs were performed during first phase, with 8245 newborns (87.8%) passing the screening test, while 1145 children (12.1%) presented an abnormal OAE and were included in the second screening phase. Second phase involving a repeat OAE examination performed on 1077 newborns (94% of abnormal stage I subjects). In this second phase, 941 newborns (87.3%) passed the test and 136 newborns (12.6%) failed. These 136 neonates were further taken up for the stage 3 screening using BERA. This stage also included the 181 newborns (1.8%) presented high-risk factors at birth and were also included in this third phase. Hearing loss was found in 4.5 % of study population.[3]

In our study, the high risk factors present in the 3 neonates with hearing loss were tabulated and were found to be family history of hearing loss (2 out of 3 neonates), TORCH infections (1 out of 3 neonates), low birth weight in all 3 neonates, hyperbilirubinemia in 1 neonate, use of ototoxic medications in 1 neonate, perinatal asphyxia in 2 neonates and requirement of mechanical ventilation >5 days in 1 neonate.

Sr. no.	Gender	High risk factors present
1	Male	Family history of SNHL, low birth weight, hyperbilirubinemia, TORCH infections
2	Female	Low birth weight, use of ototoxic medication, perinatal asphyxia at a low APGAR at 1 min
3	Male	Family history of SNHL, low birth weight, perinatal asphyxia at a low APGAR at 1 and 5mins, requirement of mechanical ventilation for more than 5 days

In a study by Fortnum H et al, out of 556 neonates who had a congenital hearing impairment, 29% had a stay in neonatal intensive care > or = 48 hours, 30% had a family history of permanent childhood hearing impairment, and 12% had a cranio-facial abnormality (CFA).[27]

In a study conducted by Kumar A et al, they found that hearing loss is more common in babies with risk factors like NICU admission in 8 out of 11 neonates with hearing loss(72.7%), LBW in 6 neonates out of 11(54.5%) and hypoxia in 6 neonates out of 11(54.5%).[84]

In a study done by Nagapoornima et al, 38 babies with severe hyperbilirubinemia requiring exchange transfusion were screened but none had hearing impairment in contrast to our study where one of 1 neonate had a positive history of hyperbilirubinemia and required exchange transfusion.[53]

Incidence of hearing loss in our study

The overall incidence of hearing loss was 6% (3 out of 50 neonates) in our study.

In a study by Khaimook W et al the incidence of hearing loss was 0.86% which is less than our study.[77]

In another study by Abdullah A et al the incidence of hearing loss was 0.42 percent.[51]

In a study by Kamath R, the overall incidence of hearing loss was 0.24% which is less than the incidence in our study.[79]

In a study by Di Stadio A et al the incidence of hearing loss was found to be 9.1% which was higher than the incidence in our study.[78]

The incidence of hearing loss in a study conducted by Nagapoornima et al. who identified 3 out of 279 high risk babies was 1.07%.[53]

A study done by Ohl C et al. 4.55% were diagnosed to have hearing loss which is lower than our study.[85]

In a similar study done by Finckh Kramer et al. 1062 at risk neonates were screened among which 1.3% neonates were detected to have hearing loss which is lower than our study.[86]

Fakhraee et al. screened 150 high risk infants of whom 42(28%) had different levels of hearing impairment which is higher than our study.[87]

Lutman et al. studied 218 patients who were at risk. The incidence of hearing loss in neonates who were in the NICU for more than 48 hours was about 2-4%.[88]

Sr no.	Name of the study	Incidence of hearing loss (%)
1	In our study	6
2	Khaimook W et al [77]	0.86
3	Abdullah A et al [51]	0.42
4	Kamath R[79]	0.24
5	Di Stadio A et al[78]	9.1
6	Nagapoornima et al[15]	1.07
7	Ohl C et al[85]	4.55
8	Finckh Kramer et al[86]	1.3
9	Fakhraee et al[87]	28
10	Lutman et al[88]	2-4

The correlation between OAE results and variables:

We had an overall OAE results of REFER in 11 neonates in this study.

1. Age of neonate :

In our study, the overall results of OAE were REFER mostly in the age group of <7days (5 neonates, 45.5% of all neonates with results as

REFER). No statistical significance could be seen between the age of neonates and outcomes of OAE screening.

In a study by Kamath R, no statistical significance was seen between the age of neonates and the outcomes of the study.[79]

2. Gender :

In our study, more males (7 neonates, 63.6%) had overall results as REFER as compared to females (4 neonates, 36.4%). No statistical significance could be seen between the gender of neonates and outcomes of OAE screening.

Similar results were seen in a study by Raghuwanshi SK et al as there was no statistical significance between the gender of neonate and the outcomes of the screening.[4]

In another study by Kaveh M et al, gender of neonate had no statistical significance with the study outcome.[14]

3. Age of parents:

In our study, the mean age of fathers was 27.24 years and of mothers was 25.98 years. No statistical significance could be seen between the age of parents and outcomes of OAE screening.

4. Occupation of parents, birth order and area of household:

The occupations of parents were statistically not significant when compared to outcomes of OAE screening. Also no statistical significance could be seen with area of household and birth order with the outcomes of the OAE screening, in our study.

5. APGAR score

APGAR score of 47 neonates was known at 1min and at 5 mins. The mean APGAR score at 1 min was 6.36 and at 5 mins was 8.02.

It was observed that as the APGAR score increased from 3 to 9 at 1 min, the number of neonates with OAE results as REFER decreased. It indicates that a low APGAR score can be a predictor of abnormal OAE

results. Statistically the APGAR score, when compared to OAE screening results, was significant (p value: 0.022).

A similar pattern was noted when the APGAR score at 5mins was observed. These results were also statistically significant when compared to the outcomes of the OAE screening (p value: 0.006).

Also in a study by Gouri ZU et al, 16.7 % cases out of 18 cases of hearing loss were found to have Apgar score ≤5. This was strongly associated risk factor for neonatal hearing loss as fetal hypoxia can damage neonates hearing system and lead to abnormal screening results.[89]

Similar outcomes were observed in the survey performed by Thomson et al. on newborn screening.[90] In his study 39 (1.2 %) babies of APGAR ≤5 were found to fail the screening tests.

Low Apgar score is also included as a risk factor for hearing impairment in JCIH 2000 criteria.[91] Our results thus corroborates with the JCIH criteria.

However in a study by Kaveh M et al the statistical analysis of APGAR score with the study outcomes was not significant.[14]

Also in a study by A. Suppiej et al, when comparison between low APGAR score and outcomes of the OAE screening was made, it was found to be statistically significant.[82]

Di Stadio A et al, in their study concluded that there was no statistical significance between the study outcomes and APGAR score of the newborn.[78]

6. Maternal antenatal history:

We observed that out of the neonates with overall OAE results as REFER, 1(9.1%) mothers had a history of hypertension, 3(27.3%) had a history of preeclampsia, 1(9.1%) had history of diabetes,1 had history of sensorineural hearing loss and none had a history of thyroid disorders and epilepsy.

None of the antenatal factors were statistically significant with the outcomes of OAE.

In a study by Gouri ZU et al, most of the antenatal risk factor for hearing loss were found non-significant except the history of antepartum hemorrhage and history of antenatal blood transfusion in the mother. They concluded that apparent cause of these antenatal risk factors associated with hearing loss may be due to intrauterine distress to fetus followed by birth asphyxia.[89]

7. Place and Type of delivery:

In our study we concluded that place of delivery and type of delivery had no statistical significance with the OAE results.

Similarly in a study by Gouri ZU et al, the method of delivery had no significant role on newborn hearing impairment.[89]

8. Family history of SNHL:

In our study, out of the 14 neonates with family history of hearing loss in childhood, 3(21.4%) had overall OAE results as REFER. This constitutes 27.3% of total neonates(11) with results as REFER. Correlation of family history of SNHL in childhood with the outcomes of OAE screening was statistically not significant.

However in a study by Salvago P et al, family history of SNHL was found to be a significant risk factor for hearing loss.[92]

Also in a study by Anand S et al, family history of hearing loss was statistitically significant with the study outcomes.[70]

9. History of TORCH infections:

In our study, out of the 8 neonates with TORCH infections, 2(25%) failed the overall OAE screening. It constitutes 18.1% of total neonates who failed the overall OAE screening. Correlation of history of TORCH infections with the outcomes of OAE screening was statistically not significant.

Similarly in a study by Elahi MM et al, history of TORCH infections in neonates was not a statistically significant factor.[93]

However in a study by Salvago P et al TORCH infections were found to be a significant risk factor for hearing loss.[92]

10. Low birth weight:

In our study, there were 39 neonates with low birth weight. Out of which, 10 (25.6%) neonates failed the overall OAE screening. This constitutes 90.9% of the total neonates with abnormal OAE screening. Correlation of low birth weight with the outcomes of OAE screening was statistically not significant.

These results were similar in a study by Ohl C et al, in which very low birth weight is not a risk factor for hearing impairment. [85]

Also a study by Finckh Kramer U et al. and Hess M et al. concluded that very low birth weight was not a predictor of hearing impairment as in our study. [84,94]

However Vohr et al in their study concluded that low birth weight was a significant high risk factor for hearing loss.[95]

11. Hyperbilirubinemia

Out of 11 neonates with overall OAE test results as REFER, 3(27.3%) showed the presence of hyperbilirubinemia. Correlation of hyperbilirubinemia with the outcomes of OAE screening was statistically not significant.

Similar results were seen in a study by Khaimook W et al, as there was no statistical significance between hyperbilirubinemia and study outcomes.[78]

Also in a study by Bergman I et al, hyperbilirubinemia was not statistically significant with the study outcomes.[96]

However in a study by Anand S et al, hyperbilirubinemia was statistitically significant with the study outcomes.[70]

12. Use of ototoxic medications:

Out of 11 neonates with overall OAE test results as REFER, history of use of ototoxic medications was seen in 1(9.1%). Correlation of use of ototoxic medications with the outcomes of OAE screening was statistically not significant

The results were contrasting in a study by Anand S et al where use of ototoxic medications was statistitically significant with the study outcomes.[70]

Similarly Vohr et al in their study concluded that use of ototoxic medications was a significant high risk factor for hearing loss.[95]

13. Need for mechanical ventilation ≥5 days:

Out of 11 neonates with overall OAE test results as REFER, history of mechanical ventilation for >5 days was observed in 5(45.5%) neonates. Correlation of need for mechanical ventilation ≥5days with the outcomes of OAE screening was statistically not significant

Similarly in a study by Di Stadio A et al., there was no statistical significance between the study outcomes and need for mechanical ventilation.[78]

However Anand S et al in their study concluded that history of mechanical ventilation for >5 days was statistitically significant with the study outcomes.[70]

Vohr et al also, in their study concluded that need for mechanical ventilation was a significant high risk factor for hearing loss.[95]

In a study by Hille ET et al, mechanical ventilation for ≥5 days was concluded to be a statistically significant factor.[41]

A 2 stage study was conducted by MD Mohd Khiari et al in which hearing assessment was done in 401 at risk neonates. He concluded that mechanical ventilation of more than 5 days was not an independent risk factor for hearing impairment as in our study.[97]

14. Craniofacial anomalies and Bacterial meningitis:

In neonates with OAE results as REFER, none had the history of craniofacial anomalies and bacterial meningitis.

15. Perinatal asphyxia at a low APGAR score:

Perinatal asphyxia however when seen at a low APGAR score (≤5 at 1min and ≤6 at 5mins) It was observed that out of 11 neonates who failed the overall OAE screening, history of perinatal asphyxia with a low APGAR at 1 min was seen in 8(72.7%) neonates and 7(63.6%) neonates had history of perinatal asphyxia at a low APGAR score at 5 mins. Correlation of perinatal asphyxia at a low apgar with the outcomes of OAE screening was statistically significant (p value 0.007 at 1 min, 0.003 at 5 mins)

Similar study conducted by Anand S et al, concluded perinatal asphyxia as a significant factor for hearing loss in newborns.[70]

Also Vohr et al in their study concluded that perinatal asphyxia was a statistically significant high risk factor for hearing loss.[95]

In a study by Hille ET et al, perinatal asphyxia was concluded to be a statistically significant factor.[41]

However in a study Charuvanij A et al, perinatal asphyxia was concluded to be not a significant high risk factor.[98]

SUMMARY & CONCLUSION

The present study on Screening for hearing loss in high risk neonates using otoacoustic emissions was a prospective observational study. It was conducted in the level III Neonatal intensive care unit (NICU) of a tertiary care teaching hospital, Dayanand Medical College and Hospital, Ludhiana by the department of Otorhinolaryngology. This surgery was in collaboration with the paediatrics team of this hospital. The study was conducted over a period of 1 year from 1st April 2021 to 31st March 2022.

We draw the following conclusions from our study:

1. The mean age of neonates in our study was 8.98 days.
2. In our study, there were 23(46%) females and 27(54%) males.
3. The following were the incidences of various high risk factors for neonatal hearing loss (as per the inclusion criteria) in our study:
    - Family history of sensorineural hearing loss : 14(28%) neonates
    - History of TORCH infections : 8(16%) neonates
    - Craniofacial anamolies : 1(2%) neonates
    - Low birth weight : 39 (78%) neonates
    - Hyperbilirubenimia : 5(10%) neonates
    - Use of ototoxic medications: 3(6%) neonates
    - Bacterial meningitis : 2(4%) neonates
    - Perinatal asphyxia at 1 min : 19(38%) neonates
    - Perinatal asphyxia at 5 mins : 14(28%) neonates
    - Need for mechanical ventilation ≥5 days : 12(24%) neonates
    - In our study there was no neonate with stigmata or syndromes associated with SNHL
4. OAE stage I results were PASS in 22 (44%) and REFER in 28 (56%) neonates in our study.
5. OAE stage II results were PASS in 17(34%) and REFER in 11 (22%) neonates in our study.

6. OAE (overall) results were PASS in 39(78%) and REFER in 11(22%) neonates in our study.
7. BERA was done in 11 neonates out which 8(16%) neonates had normal results and 3(6%) neonates had abnormal results.
8. The overall incidence of hearing loss in our study was 6%.
9. APGAR score at 1 min and at 5 mins, had a statistically significant correlation with the outcomes of OAE screening in our study. We concluded that lower APGAR scores at 1 min and at 5 mins were associated with abnormal OAE results.
10. Perinatal asphyxia at a low APGAR score at 1 min and at 5mins, had a statistically significant correlation with the outcomes of OAE screening in our study. Thus implying that perinatal asphyxia is also associated with abnormal OAE results.
11. Low birth weight, history of TORCH infections, hyperbilirubinemia, craniofacial anomalies, family history of SNHL in childhood, use of ototoxic medications and need for mechanical ventilation ≥5 days had no statistically significant correlation with the outcomes of OAE screening in our study.

BIBLIOGRAPHY

1. World Health Organization. Primary ear and hearing care training resource. World Health Organization; 2006.
2. Graven SN, Browne JV. Auditory development in the fetus and infant. Newborn and infant nursing reviews. 2008;8:187-93.
3. Escobar-Ipuz FA, Soria-Bretones C, García-Jiménez MA, Cueto EM, Aranda AM, Sotos JM. Early detection of neonatal hearing loss by otoacoustic emissions and auditory brainstem response over 10 years of experience. International journal of pediatric otorhinolaryngology. 2019;127:109647.
4. SK, Gargava A, Kulkarani V, Kumar A. Role of otoacoustic emission test in neonatal screening at tertiary center. Indian Journal of Otolaryngology and Head & Neck Surgery. 2019;71:1535-7.
5. WHO M. WHO global estimates on prevalence of hearing loss. Mortality and Burden of Diseases and Prevention of Blindness and Deafness WHO; 2012.
6. Resnikoff S, Pascolini D, Etya'ale D, Kocur I, Pararajasegaram R, Pokharel GP, et al. Global data on visual impairment in the year 2002. Bull World Health Organ. 2004;82:844-51.
7. Markides A. Shortpaper: Age at fitting of hearing aids and speech intelligibility. British journal of audiology. 1986;20:165-7.
8. World Health Organization. Newborn and infant hearing screening: Current issues and guiding principles for action. Geneva: WHO. 2010.
9. American Academy of Pediatrics Joint Committee on Infant hearing: Position statement 1982. Pediatrics. 1982;70:496-7.
10. Sancho J, Hughes E, Davis A, Haggard MP. Epidemiology of hearing impairments in children and its application to screening. Pediatric Audiology 0-5 Years. Taylor and Francis, London; 1988. p. 1-35.
11. Early identification of hearing impairment in infants and young children. NIH Consens Statement. 1993;11:1-24.

12. Dhingra PL, Dhingra S. Diseases of Ear, nose and throat & Head and Neck surgery 7th edition; 2018.
13. Watkinson JC, Clark RW, Clark LJ, Mehanna HM, Donne AJ, McGarry GW, et al (editors). Scott Brown's Otorhinolaryngology Head and Neck Surgery. Basic Sciences Endocrine Surgery Rhinology. 8th edition. CRC Press: US; 2018. p. 1081-106
14. Kaveh M, Mirjalali SN, Shariat M, Zarkesh MR. Perinatal factors influencing the neonatal hearing screening results. BMC Pediatr. 2021;21:15.
15. Kemp DT, Ryan S. Otoacoustic emission tests in neonatal screening programmes. Acta Otolaryngol Suppl. 1991;482:73-84.
16. Lieu JE, Kenna M, Anne S, Davidson L. Hearing loss in children: a review. Jama. 2020;324:2195-205.
17. Kliegman RN, Stanton BF, St Geme JM, Schor NF, Behrman RE, (editors) Nelson textbook of Pediatrics 21st edition, Philadelphia: Elsevier; 2016.
18. "Joint Committee on Infant Hearing". Retrieved 2019-03-01.
19. Foust T, Eiserman W, Shisler L, Geroso A. Using otoacoustic emissions to screen young children for hearing loss in primary care settings. Pediatrics. 2013;132:118-23.
20. Steel KP, Kros CJ. A genetic approach to understanding auditory function. Nature genetics. 2001;27:143-9.
21. Rehm HL. A genetic approach to the child with sensorineural hearing loss. In: Rehm HL (editor).Seminars in perinatology, WB Saunders. Vol 29; 2005 p. 173-81.
22. De Leenheer EM, Janssens S, Padalko E, Loose D, Leroy BP, Dhooge IJ. Etiological diagnosis in the hearing impaired newborn: proposal of a flow chart. International journal of pediatric otorhinolaryngology. 2011;75:27-32
23. Vos B, Senterre C, Lagasse R, Levêque A. Newborn hearing screening programme in Belgium: a consensus recommendation on risk factors. BMC pediatrics. 2015;15:1-4.
24. Leal MC, Muniz LF, Ferreira TS, Santos CM, Almeida LC, Linden VV, et al. Hearing loss in infants with microcephaly and evidence of

congenitalZika virus infection—Brazil, November 2015–May 2016. Morbidity and Mortality Weekly Report. 2016;65:917-9.

25. Kennedy CR, Kimm L, Dees DC. Controlled trial of universal neonatal screening for early identification of permanent childhood hearing impairment. Lancet. 1998;352:1957–64.
26. Davis A, Wood S. The epidemiology of childhood hearing impairment: factors relevant to planning services. Br J Audiol. 1992;26:77–90.
27. Fortnum H, Davis A. Epidemiology of permanent childhood hearing impairment in Trent region, 1985–1993. Br J Audiol. 1997;31:409–46.
28. Fortnum HM, Summerfield AQ, Marshall DH, et al. Prevalence of permanent childhood hearing impairment in the United Kingdom and implications for universal neonatal hearing screening: questionnaire based ascertainment study. BMJ. 2001;323:536–9.
29. Sininger YS, Doyle KJ, Moore JK. The case for early identification of hearing loss in children: auditory system development, experimental auditory deprivation, and development of speech perception and hearing. Pediatric Clinics of North America. 1999;46:1-4
30. Cristobal R, Oghalai JS. Hearing loss in children with very low birth weight: current review of epidemiology and pathophysiology. Archives of Disease in Childhood Fetal and Neonatal Edition. 2008;93:F462-8.
31. American Academy of Audiology. Zika virus disease outbreak andinfant hearing loss. 2016. http://www.audiology.org/news/zika-virus-disease-outbreak-and-infanthearing-l
32. Committee on Environmental Health. Noise: a hazard for the fetus and newborn. Pediatrics. 1997;100:724-7.
33. Roush P, Frymark T, Venediktov R, Wang B. Audiologic management of auditory neuropathy spectrum disorder in children: a systematic review of the literature. American Journal of Audiology. 2011;20:159-70.

34. Pham NS. The management of pediatric hearing loss caused by auditory neuropathy spectrum disorder. Current opinion in otolaryngology & head and neck surgery. 2017;25:396-9
35. Cristobal R, Oghalai JS. Hearing loss in children with very low birth weight: current review of epidemiology and pathophysiology. Archives of Disease in Childhood Fetal and Neonatal Edition. 2008;93:F462-8.
36. Fausti SA, Henry JA, Helt WJ, Phillips DS, Frey RH, Noffsinger D, Larson VD, Fowler CG. An individualized, sensitive frequency range for early detection of ototoxicity. Ear and hearing. 1999;20:497-505.
37. Williams AL, van Drongelen W, Lasky RE. Noise in contemporary neonatal intensive care. The Journal of the Acoustical Society of America. 2007;121:2681-90.
38. Ranganna R, Bustani P. Reducing noise on the neonatal unit. Infant. 2011;7:25
39. Shapiro SM, Popelka GR. Auditory impairment in infants at risk for bilirubin induced neurologic dysfunction. Seminars in perinatology. 2011;35:162-70.
40. Shapiro SM, Nakamura H. Bilirubin and the auditory system. Journal of Perinatology. 2001;21:S52-5.
41. Hille ET, Van Straaten HL, Verkerk PH, Dutch NICU Neonatal Hearing Screening Working Group. Prevalence and independent risk factors for hearing loss in NICU infants. Actapaediatrica. 2007;96:1155-8
42. Liden G, Kankkunen A. Visual reinforcement audiometry. Acta Otolaryngol 1969;67:281–92.
43. Ewing IR, Ewing AWG. The ascertainment of deafness in infancy and early childhood. J Laryngol Otol. 1947;59:309–38
44. Ching, Teresa Y C, and Greg Leigh. “Considering the impact of Universal Newborn Hearing Screening and early intervention on language outcomes for children with congenital hearing loss.” Hearing, balance and communication. 2020;18:215-24.
45. Ewing AWG, Ewing EC. Hearing-impaired children under five: a guide for parents and teachers [by] Sir Alexander Ewing & Lady

Ethel C. Ewing Manchester University Press; Volta Bureau Manchester, Washington, D.C; 1971.

46. Wedenberg E. Auditory tests on newborn infants". ActaOto-Laryngologica. 1956;46:446–61.
47. Downs MP, Sterritt GM. A guide to new born and infant hearing screening programs. Arch Otolaryngol Head Neck Surg. 1967;85:37–44.
48. Northern JL, Downs MP. Hearing in Infants. 5th ed. Baltimore, MD:Lippincott Williams & Wilkins; 2002
49. Universal hearing screening in Illinois 2003-2004, published report of Illinois Dept of Public health, Division of Epidemiologic studies. 2006;1-11.
50. White KR, Maxon AB. Universal screening for infant hearing impairment: simple, beneficial, and presently justified. International journal of pediatric otorhinolaryngology. 1995;32:201-10.
51. Abdullah A, Hazim MY, Almyzan A, Jamilah AG, Roslin S, Ann MT, et al. Newborn hearing screening: experience in a Malaysian hospital. Singapore medical journal. 2006;47:60.
52. Asma A, Wan Fazlina WH, Almyzan A, Han YS, Jamilah AG, Roslin S. Benefit and pitfalls of newborn hearing screening. Med J Malaysia. 2008;63:293-7.
53. Nagapoornima P, Ramesh A, Srilakshmi, Rao S, Patricia PL, Gore M, Dominic M, et al. Universal hearing screening. Indian J Pediatr. 2007;74:545-9.
54. Ansari MS. Screening programme for hearing impairment in newborns: A challenge during rehabilitation for all. Asia pacificdisability rehabilitation journal. 2004;15:83-9.
55. Downs MP, Yoshinaga-Itano C. The efficacy of early identification and intervention for children with hearing impairment. Pediatric Clinics. 1999;46:79-87.
56. Meadow KP. Deafness and child development. University of California Press; 2022.
57. White SJ, White EC. The effects of hearing status of the family and age of intervention on receptive andexpressive oral language skills

in hearing-impaired infants. In: Levitt H, McGarr N, Geffner D, editors. Development of language and communication skillsin hearing children: ASHA monographs 26.Rockville (MD): American Speech-Language-Hearing Association; 1987. p. 25–35.

58. From National Consultation Meeting for Developing IAP Guidelines on Neurodevelopmental Disorders under the aegis of IAP Childhood Disability Group and the Committee on Child Development and Neurodevelopmental Disorders; Paul A, Prasad C, Kamath SS, Dalwai S, C Nair MK, Pagarkar W. Consensus Statement of the Indian Academy of Pediatrics on Newborn Hearing Screening. Indian Pediatr. 2017;54:647-51.
59. British Society of Audiology. NHSP audiology protocols and guidance. Available from: http://www.thebsa.org.uk/resources/.
60. Public Health England. Newborn hearing screening care pathways; 2015. Available from: https://www.gov.uk/government/publications/newborn-hearing-screening-care-pathways.
61. Jones AT. The discovery of difference tones. American Journal of Physics. 1935;3:49-51.
62. Hiebert E. Physiological acoustics and combination tones. In: The Helmholtz Legacy in Physiological Acoustics. Springer, Cham; 2014. p. 33-8.
63. Sumitrajit D, Hall JW, Wilbur J. Otoacoustic Emissions Principles, Procedures, and Protocols Second Edition; 2018.
64. Meyer MF. Subjective Tones: Tartini and Beat-Tone Pitches. The American Journal of Psychology. 1957;70:646-50.
65. Caselli G, Cecchi G, Malacarne M, Masetti G. Analysis of violin combination tones and their contribution to Tartini's third tone. Savart Journal; 2020.
66. Gold T. Hearing. The physical basis of the action of the cochlea. Proceedings of the Royal Society of London. Series B-Biological Sciences. 1948;135:492-8.
67. Johnson JL, White KR, Widen JE, Gravel JS, James M, Kennalley T, et al. A multicenter evaluation of how many infants with

permanent hearing loss pass a two-stage otoacoustic emissions/automated auditory brainstem response newborn hearing screening protocol. Pediatrics. 2005;116:663-72.

68. Stevens J, Foster M, Brennan S. A Comparison of ASSR and Tonepip ABR With Equivalent Test Parameters. Presented at NHS 2004: The International Conference on Newborn Hearing Screening, Diagnosis, and Intervention; 2004.
69. Stevens JC, Webb HD, Hutchinson J, Connell J, Smith MF, Buffin JT. Click evoked otoacoustic emissions in neonatal screening. Ear and hearing. 1990;11:128-33.
70. Anand S, Tiwari A, Goyal S. Prospective study for newborn hearing screening-A experience from tertiary care centre in central India. Int J Pediatr Res. 2016;3:668-71.
71. Rughani S, Vyas B, Sinha V. Hearing screening in newborns (a cross sectional study), WAENT.com; 2011. Available from: https://www.entworld.org/index.php/journal-archives/combined/702-hearing-screening-in-newborns-a-cross-sectional-study
72. Bhatt J, Chhangte L. Accuracy of OAE and BERA to detect the incidence of hearing loss in newborn. International Journal of Scientific and Research Publications. 2015;5:1-6.
73. Heinemann M, Bohnert A. Hearing screening in newborn infants. Comparative studies and cost analysis with different instruments. Laryngo-rhino-otologie. 2000;79:453-8.
74. Mishra G, Sharma Y, Mehta K, Patel G. Efficacy of Distortion Product Oto-Acoustic Emission (OAE)/Auditory Brainstem Evoked Response (ABR) protocols in universal neonatal hearing screening and detecting hearing loss in children< 2 years of age. Indian Journal of Otolaryngology and Head & Neck Surgery. 2013;65:105-10.
75. Keren R, Helfand M, Homer C, McPhillips H, Lieu TA. Projected cost-effectiveness ofstatewide universal newborn hearing screening. Pediatrics. 2002;110:855-64.
76. Roizen NJ. Etiology of hearing loss in children: nongenetic causes. Pediatric Clinics of North America. 1999;46:49-64.

77. Khaimook W, Chayarpham S, Dissaneevate S. The high-risk neonatal hearing screening program in Songklanagarind Hospital. Medical journal of the Medical Association of Thailand. 2008;91:1038.
78. Di Stadio A, Dipietro L, Ralli M, Meneghello F, Minni A, Greco A, Stabile MR, Bernitsas E. Sudden hearing loss as an early detector of multiple sclerosis: a systematic review. Eur Rev Med Pharmacol Sci. 2018;22:4611-24.
79. Kamath R. Hearing screening for babies admitted to NICU. SDM college of medical sciences and hospital (Doctoral dissertation, Shri Dharmasthala Manjunatheshwara University, Dharwad); 2022. Available from: https://scholar.google.com/scholar?hl=en&as_sdt=0%2c5&q=hearing+screening+for+babies+admitted+to+nicu+at+sdm+college+of+medical+sciences+and+hospital%e2%80%9d&btng=
80. Zhu Y, Hu L, Yang L, Wang L, Lu Y, Dong X, et al. Association between expanded genomic sequencing combined with hearing screening and detection of hearing loss among newborns in a neonatal intensive care unit. JAMA network open. 2022;5:e2220986.
81. De Kock T, Swanepoel D, Hall III JW. Newborn hearing screening at a community-based obstetric unit: Screening and diagnostic outcomes. International Journal of Pediatric Otorhinolaryngology. 2016;84:124-31.
82. Suppiej A, Rizzardi E, Zanardo V, Franzoi M, Ermani M, Orzan E. Reliability of hearing screening in high-risk neonates: comparative study of otoacoustic emission, automated and conventional auditory brainstem response. Clinical Neurophysiology. 2007;118:869-76.
83. Sachdeva K, Sao T. Outcomes of Newborn Hearing Screening Program: A Hospital Based Study. Indian J Otolaryngol Head Neck Surg. 2017;69:194-8.
84. Kumar A, Shah N, Patel KB, Vishwakarma R. Hearing screening in a tertiary care hospital in India. Journal of Clinical and Diagnostic Research: JCDR. 2015;9:MC01.

85. Ohl C, Dornier L, Czajka C, Chobaut JC, Tavernier L. Newborn hearing screening on infants at risk. International journal of pediatric otorhinolaryngology. 2009;73:1691-5.
86. Sharma P, Chhangani NP, Meena KR, Jora R, Sharma N, Gupta BD. Brainstem evoked response audiometry (BAER) in neonates with hyperbilirubinemia. The Indian Journal of Pediatrics. 2006;73:413-6
87. Yoshinaga-Itano C, Sedey AL, Coulter DK, Mehl AL. Language of early-and later identified children with hearing loss. Pediatrics. 1998;102:1161-71.
88. Nadaraja GS, Gurgel RK, Kim J, Chang KW. Hearing outcomes of atresia surgery versus osseointegrated bone conduction device in patients with congenital aural atresia: a systematic review. Otology & Neurotology. 2013;34:1394-9.
89. Gouri ZU, Sharma D, Berwal PK, Pandita A, Pawar S. Hearing impairment and its risk factors by newborn screening in north-western India. Maternal health, neonatology and perinatology. 2015;1:1-8.
90. John M, Balraj A, Kurien M. Neonatal screening for hearing loss: pilot study from a tertiary care centre. Indian J Otolaryngol Head Neck Surg. 2009;61:23–6.
91. Joint Committee on Infant Hearing. Year 2000 position statement: principles and guidelines for early hearing detection and intervention programs. Pediatrics. 2000;106:798–817.
92. Salvago P, Martines E, Martines F. Prevalence and risk factors for sensorineural hearing loss: Western Sicily overview. European Archives of Oto-Rhino-Laryngology. 2013;270:3049-56.
93. Elahi MM, Elahi F, Elahi A, Elahi SB. Paediatric hearing loss in rural Pakistan. Journal of Otolaryngology-Head & Neck Surgery. 1998;27:348.
94. Hess M, Finckh-Krämer U, Bartsch M, Kewitz G, Versmold H, Gross M. Hearing screening in at-risk neonate cohort. International journal of paediatrics otorhinolaryngology. 1998;46:81-9.

95. Vohr BR, Widen JE, Cone-Wesson B, Sininger YS, Gorga MP, Folsom RC, et al. Identification of neonatal hearing impairment: characteristics of infants in the neonatal intensive care unit and well-baby nursery. Ear Hear. 2000;21:373-82.
96. Bergman I, Hirsch RP, Fria TJ, Shapiro SM, Holzman I, Painter MJ. Cause of hearing loss in the high-risk premature infant. The Journal of pediatrics. 1985;106:95-101.
97. Ahmad A, Mohamad I, Mansor S, Daud MK, Sidek D. Outcome of a newborn hearing screening program in a tertiary hospital in Malaysia: the first five years. Ann Saudi Med. 2011;31:24-8.
98. Charuvanij A, Visudhiphan P, Chiemchanya S, Tawin C. Sensorineural hearing loss in children recovered from purulent meningitis: a study in Thai children at Ramathibodi Hospital. J Med Assoc Thai. 1990;73:253-7.

S.No.	MRD No.	Age (Days)	Gender	Age of parents (YEARS)		Parental occupation		town/village	urban/rural	Birth Order	APGAR score		ANTENATAL HISTORY				
				Mother	Father	Mother	Father				At 1 min	At 5 mins	H/O Hypertension	H/o preeclampsia	h/o Diabetes	H/o thyroid	H/o Epilepsy
1	676559	2	F	26	28	HOUSEWIFE	BUSINESSMAN	TOWN	URBAN	2	7	9	N	N	N	Y (OVERT)	N
2	1133577	1	M	23	25	HOUSEWIFE	SHOPKEEPER	TOWN	URBAN	1	8	9	N	Y	N	N	N
3	1124461	1	F	26	26	HOUSEWIFE	SHOPKEEPER	TOWN	URBAN	1	5	8	Y	Y	N	N	N
4	1123919	25	M	28	28	HOUSEWIFE	PRIVATE JOB	TOWN	URBAN	1	3	6	Y	Y	N	N	N
5	1122243	5	F	27	27	HOUSEWIFE	BUSINESSMAN	VILLAGE	RURAL	1	5	7	N	N	N	Y (OVERT)	N
6	1132922	4	M	25	26	HOUSEWIFE	EVENT ORGANISER	TOWN	URBAN	2	8	8	N	N	N	N	N
7	1131477	8	M	28	28	TEACHER	MARKETING	TOWN	URBAN	1	7	8	N	N	N	N	N
8	1116261	5	M	23	25	WORKER	WORKER	TOWN	URBAN	1	6	7	Y	N	N	N	N
9	1138179	1	M	27	27	TEACHER	BUSINESSMAN	TOWN	URBAN	1	8	9	N	N	N	Y (OVERT)	N
10	1133564	16	M	29	29	HOUSEWIFE	SHOPKEEPER	TOWN	URBAN	2	5	6	Y	N	N	N	N
11	1138279	28	M	23	25	HOUSEWIFE	SHOPKEEPER	VILLAGE	RURAL	1	5	7	N	N	N	N	N
12	1150004	5	M	25	27	HOUSEWIFE	SHOPKEEPER	TOWN	URBAN	2	5	8	N	N	N	N	N
13	1141812	1	M	24	25	HOUSEWIFE	SERVICE	VILLAGE	RURAL	1	7	8	N	Y	N	N	N
14	1141762	25	F	25	26	HOUSEWIFE	BUSINESSMAN	VILLAGE	RURAL	2	5	7	N	Y	N	N	N
15	1167568	26	F	25	26	TEACHER	TEACHER	TOWN	URBAN	2	NK	NK	N	N	N	N	N
16	1167567	28	F	24	25	HOUSEWIFE	SERVICE	TOWN	URBAN	1	7	8	N	N	N	N	N
17	1180718	14	F	22	24	HOUSEWIFE	BUSINESSMAN	TOWN	URBAN	2	3	5	N	N	N	N	N
18	1194339	1	F	27	29	BAKER	SHOPKEEPER	TOWN	URBAN	1	9	10	N	N	N	Y (OVERT)	N
19	1184429	6	M	36	38	HOUSEWIFE	BUSINESSMAN	TOWN	URBAN	3	5	7	N	Y	N	N	N
20	1243553	1	F	28	28	HOUSEWIFE	BUINESSMAN	TOWN	URBAN	2	8	9	Y	N	N	N	N
21	1324570	2	M	25	28	HOUSEWIFE	BUSINESSMAN	VILLAGE	RURAL	1	8	9	N	N	N	N	N
22	1317430	3	F	24	24	HOUSEWIFE	BUSINESSMAN	VILLAGE	RURAL	1	6	8	N	N	N	N	N
23	1370881	3	F	26	28	TEACHER	SERVICE	VILLAGE	RURAL	1	3	5	Y	N	N	N	N
24	1301148	6	M	27	28	HOUSEWIFE	BUSINESSMAN	TOWN	URBAN	1	3	5	N	N	N	N	N
25	1338707	10	F	28	28	HOUSEWIFE	SERVICE	VILLAGE	RURAL	1	8	9	N	N	N	N	N
26	1336535	3	M	26	28	HOUSEWIFE	BUSINESSMAN	TOWN	URBAN	1	8	10	N	N	N	N	N
27	1239824	1	M	26	28	DOCTOR	DOCTOR	TOWN	URBAN	1	8	10	N	N	N	N	N
28	1283298	19	F	26	27	HOUSEWIFE	SERVICE	VILLAGE	RURAL	1	8	10	N	N	N	N	N
29	1350175	2	M	28	28	TEACHER	SERVICE	TOWN	URBAN	1	8	9	N	N	N	Y (OVERT)	N
30	1358823	20	F	25	28	HOUSEWIFE	BUSINESSMAN	TOWN	URBAN	1	8	9	N	N	Y	N	N
31	1305200	3	F	28	30	HOUSEWIFE	FARMER	VILLAGE	RURAL	2	4	6	N	N	N	N	N
32	1350091	8	M	23	28	HOUSEWIFE	SHOPKEEPER	VILLAGE	RURAL	2	NK	NK	N	N	N	N	N
33	1334817	8	F	26	28	HOUSEWIFE	SERVICE	TOWN	URBAN	2	8	10	N	N	N	N	Y
34	1365238	2	M	33	35	TEACHER	BUSINESSMAN	TOWN	URBAN	3	7	9	N	N	N	N	N
35	1383912	20	F	28	28	HOUSEWIFE	SHOPKEEPER	VILLAGE	RURAL	2	7	8	N	N	N	N	N
36	1383907	20	M	21	23	HOUSEWIFE	FARMER	VILLAGE	RURAL	1	4	6	N	N	N	N	N
37	1388966	9	M	24	25	DOCTOR	DOCTOR	TOWN	URBAN	1	7	8	N	N	N	N	N
38	1289844	5	F	25	27	HOUSEWIFE	LABOURER	VILLAGE	RURAL	4	NK	NK	NK	NK	NK	NK	NK
39	1392246	25	F	23	23	HOUSEWIFE	SHOPKEEPER	TOWN	URBAN	1	6	9	N	N	Y	N	N
40	1267379	2	M	24	28	HOUSEWIFE	BUSINESSMAN	TOWN	URBAN	1	6	9	N	N	N	N	N
41	1483910	11	M	26	26	HOUSEWIFE	SERVICE	TOWN	URBAN	2	8	9	N	N	N	N	N
42	1387581	2	M	28	28	HOUSEHELPER	FACTORY WORKER	TOWN	URBAN	2	7	8	N	N	N	N	N
43	1482320	10	F	23	25	STUDENT IN COLLEGE	JOB	TOWN	URBAN	1	7	10	N	N	N	N	N
44	1384201	20	M	26	26	HOUSEWIFE	BUSINESSMAN	TOWN	URBAN	1	8	10	N	N	N	N	N
45	1432210	18	F	28	28	DOCTOR	ENGINEER	TOWN	URBAN	3	7	8	N	N	N	N	N
46	1130677	3	M	25	26	SERVICE	SERVICE	TOWN	URBAN	2	7	8	N	N	N	N	N
47	1432010	8	F	26	29	HOUSEWIFE	TAILOR	TOWN	URBAN	2	5	6	N	N	Y	N	N
48	1416320	16	M	29	29	SERVICE	BUSINESSMAN	TOWN	URBAN	2	7	9	N	N	N	N	N
49	1367133	9	M	23	25	HOUSEWIFE	SHOPKEEPER	VILLAGE	RURAL	1	3	5	N	N	N	N	N
50	1389681	2	F	28	28	TEACHER	CLERK	TOWN	URBAN	2	7	9	N	N	N	N	N

S.No.	MRD No.	H/o Sensorineural hearing loss	H/o maternal drug intake	BIRTH HISTORY							
				Place of Delivery	Type of Devilery	Asphyxia/other problems	Birth Weight IN GRAMS	Gestation IN WEEKS	Family history of SNHL)	TORCH Infections)	craniofacial abnormalities
1	676559	N	THYROXINE	HOSPITAL	LSCS	LBW	2020	31+3	N	N	N
2	1133577	N	N	HOSPITAL	LSCS	LBW, EARLY ONSET SEPSIS, TCP,HYPERBILIRUBENEMIA	1520	33	Y	Y	N
3	1124461	N	N	HOSPITAL	LSCS	LBW,RDS	1740	31+3	N	N	N
4	1123919	N	N	HOSPITAL	LSCS	NO CRY,LBW, ASPHYXIA, CYANOSIS	800	32+2	N	Y	N
5	1122243	N	THYRONORM 125	HOSPITAL	LSCS	ASPHYXIA, NO CRY	950	29+5	Y	N	N
6	1132922	N	N	HOSPITAL	LSCS	NO ASPHYXIA, PATHOGICAL JAUNDICE	3400	38+1	Y	N	N
7	1131477	N	N	HOSPITAL	NVD	LBW	1640	31	N	N	Y
8	1116261	N	N	HOSPITAL	LSCS	ASPHYXIA ,LBW,OLIGOHYDROAMNIOS, LOOP AROUND NECK	940	29+3	N	Y	N
9	1138179	N	THYROXINE 50MCG	HOSPITAL	LSCS	LBW	1560	32	N	N	N
10	1133564	N	N	HOSPITAL	LSCS	ASPHYXIA	1380	31	Y	N	N
11	1138279	N	N	HOSPITAL	LSCS	LBW, ASPHYXIA	1900	35	Y	N	N
12	1150004	N	N	HOSPITAL	LSCS	LBW,RESPIRATORY DISTRESS	1280	31	N	N	N
13	1141812	N	N	HOSPITAL	LSCS	OLIGOHYDROANIOS, TOXOPLASMOSIS, PPROM	1020	28	N	Y TOXOPLASMOSIS)	N
14	1141762	N	N	HOSPITAL	LSCS	ASPHYXIA ,LBW,OLIGOHYDROAMNIOS, LOOP AROUND NECK	1800	30	N	N	N
15	1167568	N	N	HOSPITAL	LSCS	SGA, LBW	1575	34	N	N	N
16	1167567	N	N	HOSPITAL	LSCS	PTLP	1960	34	N	N	N
17	1180718	N	N	HOSPITAL	LSCS	ASPHYXIA	1270	34	N	N	N
18	1194339	N	N	HOSPITAL	LSCS	LBW	2270	33 +3	N	N	N
19	1184429	N	Y RADIATION EXPOSURE	HOSPITAL	LSCS	ELBW, RDS	980	30	N	N	N
20	1243553	N	N	HOSPITAL	LSCS	PRETERM,LBW	1460	35+1	N	N	N
21	1324570	N	N	HOSPITAL	LSCS	NO	3460	38	Y	N	N
22	1317430	N	N	HOSPITAL	LSCS	LBW, PREMATURE	2000	32+1	N	N	N
23	1370881	N	Y AMLONG	HOSPITAL	NVD	ASPHYXIA	2700	32	N	N	N
24	1301148	N	N	HOSPITAL	LSCS	PRETERM,VLBW,ASPHYXIA, POST CPR SURVIVOR	1200	29+3	N	N	N
25	1338707	Y	N	HOSPITAL	LSCS	LBW,RDS	1680	35+3	Y	N	N
26	1336535	N	N	HOSPITAL	LSCS	OLIGOHYDROAMNIOS, LBW	2040	34+2	N	N	N
27	1239824	N	N	HOSPITAL	LSCS	NONE	2900	39+3	Y	N	N
28	1283298	N	N	HOSPITAL	LSCS	SEIZURE ON DAY 9	2500	36+4	N	N	N
29	1350175	N	N	HOSPITAL	LSCS	IUGR	2080	36+3	N	N	N
30	1358823	N	ANTI DIABETIC MEASURI	HOSPITAL	LSCS	SEPTICEMIA	1900	33	N	Y (CMV AND RUBELLA)	N
31	1305200	N	N	HOSPITAL	LSCS	ASPHYXIA WITH DESATURATION WITH NEONATAL HYPERBILIRUBENIMA	2900	39	N	N	N
32	1350091	N	N	HOSPITAL	LSCS	ASPHYXIA	2400	36	N	N	N
33	1334817	N	Y ANTI EPILEPTICS	HOSPITAL	LSCS	LBW, PRETERM	1900	29+6	N	N	N
34	1365238	N	N	HOSPITAL	NVD	IMPERFORATE ANUS	3500	39+2	Y	N	N
35	1383912	N	N	HOSPITAL	LSCS	LBW, PRETERM	2000	30+6	N	N	N
36	1383907	Y	N	HOSPITAL	LSCS	LBW, PRETERM, ASPHYXIA	1500	24	Y	N	N
37	1388966	N	N	HOSPITAL	NVD	LBW	1800	32+2	Y	N	N
38	1289844	NK	N	HOME	NVD	RDS, LBW	1700	32	N	Y (RUBELLA)	N
39	1392246	N	N	HOSPITAL	LSCS	RDS, LBW	2000	34	N	N	N
40	1267379	N	Y ANTIBIOTIC FOR UTI	HOSPITAL	NVD	LBW,TCP	1900	37+2	N	Y	N
41	1483910	N	N	HOSPITAL	LSCS	LBW	2000	32	Y	N	N
42	1387581	Y	N	HOSPITAL	NVD	NO	2800	37	Y	N	N
43	1482320	N	N	HOSPITAL	LSCS	LBW , PRETERM	1200	30	N	N	N
44	1384201	N	N	HOSPITAL	LSCS	PRETERM	2100	33	N	N	N
45	1432210	N	N	HOSPITAL	LSCS	TORCH WITH CMV POSITIVE	2600	35	N	Y (CMV)	N
46	1130677	Y	Y (AMIKACIN FOR FEVER)	HOSPITAL	NVD	FEVER	3000	37	N	N	N
47	1432010	N	ANTIDIABETIC MEASURE	HOSPITAL	LSCS	ASPHYXIA	1750	32	N	N	N
48	1416320	N	N	HOSPITAL	NVD	NONE	1200	28	Y	N	N
49	1367133	N	N	HOSPITAL	NVD	LBW, TERM,IUGR ,RDS	2200	38	N	N	N
50	1389681	N	N	HOSPITAL	NVD	HYPERBILRUBENEMIA	2000	35	N	N	N

		HIGH RISK FACTOR								GPE	VITALS (At the time of testing)			
S.No.	MRD No.	low birth weight	hyperbilirubinemia	ototoxic medications	bacterial meningitis	Perinatal Asphyxia at a low APGAR		Mechanical Ventilation >5days	stigmata or others causing snhl	NORMAL (Y/N)	HR	TEMPERATURE	SPO2 (%)	EAR
						At 1 min	At 5 mins							
1	676559	Y	N	N	N	N	N	N	N	Y	118	98.3 F	97%	NORMAL
2	1133577	Y	Y	N	N	N	N	N	N	N (PALLOR)	120	98.5 F	98%	NORMAL
3	1124461	Y	Y	N	N	Y	N	N	N	Y	120	98.4 F	95%	NORMAL
4	1123919	Y	N	N	N	Y	Y	Y	N	N (PALLOR)	130	98.6F	94%	NORMAL
5	1122243	Y	N	N	N	Y	Y	Y	N	N (PALLOR)	120	99F	98	NORMAL
6	1132922	N	Y	N	N	N	N	N	N	N(ICTERUS)YELLOWISH SKIN DISCOLORATION	120	98.8 F	94	NORMAL
7	1131477	Y	N	N	N	N	N	N	N	N	140	98.6 F	96	NORMAL
8	1116261	Y	N	N	Y	Y	Y	Y	N	N(PALLOR)	132	98.7 F	95	NORMAL
9	1138179	Y	N	N	N	N	N	N	N	Y	110	98.8 F	98	NORMAL
10	1133564	Y	N	N	N	Y	Y	Y	N	Y	154	98.9 F	98	NORMAL
11	1138279	Y	N	N	N	Y	N	N	N	Y	100	99.4 F	96	NORMAL
12	1150004	N	N	N	N	Y	N	Y	N	Y	123	98.9 F	95	NORMAL
13	1141812	Y	N	N	N	N	N	N	N	Y	130	98.5 F	98	NORMAL
14	1141762	Y	N	Y (AMIKACIN FOR 10DAYS)	N	Y	N	N	N	N (PALLOR)	100	99.1 F	96	NORMAL
15	1167568	Y	N	Y (VANCOMYCIN)	N	Y	N	N	N	Y	134	98.5 F	98	NORMAL
16	1167567	Y	N	N	N	N	N	N	N	Y	120	98.7 F	95	NORMAL
17	1180718	Y	N	N	N	Y	Y	Y	N	Y	132	97.8 F	98	NORMAL, WAX
18	1194339	Y	N	N	N	N	N	N	N	Y	140	98.8 F	98	NORMAL
19	1184429	Y	N	N	N	Y	Y	Y	N	N (PALLOR)	140	98.6 F	98	NORMAL
20	1243553	Y	N	N	N	N	N	N	N	Y	142	98.6 F	96	NORMAL
21	1324570	N	N	N	N	N	N	N	N	Y	138	98.3 F	95	NORMAL
22	1317430	Y	N	N	N	N	N	N	N	Y	130	98.6 F	98	NORMAL
23	1370881	N	N	N	N	Y	Y	N	N	Y	138	98.5 F	98	NORMAL
24	1301148	Y	N	N	N	Y	Y	Y	N	N (PALLOR)	110	98.8 F	95	NORMAL
25	1338707	Y	N	N	N	N	N	N	N	Y	132	98.4 F	98	NORMAL
26	1336535	Y	N	N	N	N	N	N	N	Y	150	98.6 F	96	NORMAL
27	1239824	N	N	N	N	N	N	N	N	Y	150	96.8 F	98	NORMAL
28	1283298	N	N	N	Y	N	N	N	N	Y	123	97.6 F	98	NORMAL
29	1350175	Y	N	N	N	N	N	N	N	Y	130	98.5 F	97	NORMAL
30	1358823	Y	N	N	N	N	N	N	N	Y	118	98 F	98	NORMAL
31	1305200	N	Y	N	N	Y	Y	N	N	Y	134	98.6 F	99	NORMAL
32	1350091	Y	N	N	N	Y	Y	Y	N	Y	132	98 F	98	NORMAL
33	1334817	Y	N	N	N	N	N	N	N	Y	143	99 F	94	NORMAL
34	1365238	N	N	N	N	N	N	N	N	Y	142	98.4 F	97	NORMAL
35	1383912	Y	N	N	N	N	N	N	N	Y	148	97.8 F	96	WAX B/L
36	1383907	Y	N	N	N	Y	Y	Y	N	N (PALLOR)	147	99.5 F	96	NORMAL
37	1388966	Y	N	N	N	N	N	N	N	Y	130	98.7 F	98	NORMAL
38	1289844	Y	N	N	N	Y	Y	Y	N	Y	140	97. 8 F	92	NORMAL
39	1392246	Y	N	N	N	N	N	N	N	N (PALLOR)	140	97 F	98	NORMAL
40	1267379	Y	N	N	N	N	N	N	N	N (PALLOR)	138	99.8 F	98	NORMAL
41	1483910	Y	N	N	N	N	N	N	N	N	130	98	95	NORMAL
42	1387581	N	N	N	N	N	N	N	N	N	130	99.3	95	NORMAL
43	1482320	Y	N	N	N	N	N	N	N	N	140	97.8	98	NORMAL
44	1384201	Y	N	N	N	N	N	N	N	N	130	99.4	97	NORMAL
45	1432210	N	N	N	N	N	N	N	N	N	132	98	97	NORMAL
46	1130677	N	N	Y	N	N	N	N	N	N	140	97.5	98	NORMAL
47	1432010	Y	N	N	N	Y	Y	Y	N	N	127	98.1	98	NORMAL
48	1416320	Y	N	N	N	N	N	N	N	N	129	97.5	98	NORMAL
49	1367133	Y	N	N	N	Y	Y	N	N	N	143	99.4	98	NORMAL
50	1389681	Y	Y	N	N	N	N	N	N	N	150	96.7	94	NORMAL

		LOCAL EXAMINATION		INVESTIGATIONS									TESTING		
					CSF								OAE		
S.No.	MRD No.	NOSE	THROAT	SERUM BILIRUBIN mg/dl	Protein (mg/dl)	glucose (mg/ml)	gram staining/culture sensitivity	HEMOGLOBIN (gm/dl)	TLC (10^3 /ul)	ANC (10^3/ul)	CRP (mg/dl)	BLOOD CULTURE	STAGE 1 PASS/REFER	STAGE 2	BERA
1	676559	NORMAL	NORMAL	15.6	NA	NA	NA	14.8	8	3.20	3.5	NO GROWTH	PASS	-	-
2	1133577	NORMAL	NORMAL	23.3	NA	NA	NA	17.9	7.2	3.60	0.44	NO GROWTH	REFER	REFER	hearing loss
3	1124461	NORMAL	NORMAL	25.3	NA	NA	NA	16.7	9.1	3.21	0.1	NO GROWTH	REFER	PASS	-
4	1123919	NORMAL	NORMAL	3	NA	NA	NA	7.1	12.2	4.15	0.8	NO GROWTH	REFER	PASS	-
5	1122243	NORMAL	NORMAL	8.3	NA	NA	NA	9.2	8.4	2.63	2.28	NO GROWTH	PASS	-	-
6	1132922	NORMAL	NORMAL	29.37	NA	NA	NA	14.2	12.2	6.34	0.74	NO GROWTH	PASS	-	-
7	1131477	NORMAL	NORMAL	NA	NA	NA	NA	16.5	12	7.16	0.3	NO GROWTH	PASS	-	-
8	1116261	NORMAL	NORMAL	NA	156.3	53	ENTEROCOCCUS FAECIUM	9.2	7.9	1.37	0.74	STAPHYLOCOCCUS HEMOLYTICUS	REFER	PASS	-
9	1138179	NORMAL	NORMAL	13.51	NA	NA	NA	18	7.8	5.20	0.05	NO GROWTH	PASS	-	-
10	1133564	MILD DNS TO RIGHT	TONSILLAR SWELLING	10.19	NA	NA	NA	12.1	12.4	1.36	0.26	NO GROWTH	REFER	REFER	no hearing loss
11	1138279	DNS TO RIGHT	TONSILLAR HYPERTROPHY	NA	NA	NA	NA	8	15.1	3.50	0.88	NO GROWTH	PASS	-	-
12	1150004	NORMAL	NORMAL	27.53	NA	NA	NA	14.5	20.1	16.14	2.6	NO GROWTH	PASS	-	-
13	1141812	NORMAL	NORMAL	9.1	123.1	34	no growth	11.5	1.4	0.98	35.02	NO GROWTH	REFER	PASS	
14	1141762	NORMAL	NORMAL	NA	NA	NA	no growth	10.1	9.5	2.66	2	NO GROWTH	REFER	REFER	hearing loss
15	1167568	NORMAL	TONSILLAR HYPERTROPHY	NA	NA	NA	no growth	9.2	11.1	1.67	0.66	ACINETBACTER BAUMANII	REFER	PASS	-
16	1167567	NORMAL	NORMAL	NA	NA	NA	no growth	17.1	11	8.83	3.77	NO GROWTH	PASS	-	-
17	1180718	NORMAL	NORMAL	4.8	NA	NA	NA	8.5	7	3.15	0.43	NO GROWTH	PASS	-	-
18	1194339	NORMAL	NORMAL	3.19	NA	NA	NA	14.3	7.2	3.02	0.23	NO GROWTH	PASS	-	-
19	1184429	NORMAL	NORMAL	4.25	NA	NA	NA	15.2	4.8	3.75	0.79	NO GROWTH	REFER	REFER	no hearing loss
20	1243553	NORMAL	NORMAL	7.24	NA	NA	NA	13.4	6.7	3.46	0.35	NO GROWTH	PASS	-	
21	1324570	NORMAL	NORMAL	NA	NA	NA	NA	14.2	6	2.57	0.33	NO GROWTH	REFER	PASS	-
22	1317430	NORMAL	NORMAL	9.83	NA	NA	NA	13.7	9.6	2.27	2	NO GROWTH	PASS	-	-
23	1370881	NORMAL	NORMAL	0.67	NA	NA	NA	15.4	15.9	10.64	2	NO GROWTH	PASS	-	
24	1301148	NORMAL	NORMAL	5.65	NA	NA	NA	8.2	12.6	3.30	22.8	NO GROWTH	REFER	PASS	-
25	1338707	NORMAL	NORMAL	NA	NA	NA	NA	21.9	14.7	10.10	0.67	NO GROWTH	REFER	PASS	-
26	1336535	NORMAL	NORMAL	9.9	NA	NA	NA	16.7	7.8	4.84	0.38	NO GROWTH	PASS	-	-
27	1239824	NORMAL	NORMAL	4.1	NA	NA	NA	15.8	7.2	2.88	0.4	NO GROWTH	PASS	-	-
28	1283298	NORMAL	NORMAL	3	143	28	STREPTOCOCCUS PNEUMONIAE	15.1	28.2	16.90	78.4	NO GROWTH	REFER	PASS	-
29	1350175	NORMAL	NORMAL	9.55	NA	NA	NA	21.6	25.4	10.65	1.08	NO GROWTH	PASS	-	-
30	1358823	NORMAL	NORMAL	NA	60	78	no growth	14.3	21.8	9.80	17.8	NO GROWTH	REFER	PASS	-
31	1305200	NORMAL	NORMAL	28.2	NA	NA	NA	13.5	11	5.15	13.37	NO GROWTH	REFER	REFER	no hearing loss
32	1350091	NORMAL	NORMAL	NA	NA	NA	NA	16.8	8.7	3.69	3.55	NO GROWTH	REFER	REFER	no hearing loss
33	1334817	NORMAL	NORMAL	4.8	42	95	no growth	14.8	3.8	1.50	0.33	NO GROWTH	PASS	-	-
34	1365238	NORMAL	NORMAL	0.85	NA	NA	NA	7.8	7.39	6.20	15.1	NO GROWTH	REFER	PASS	-
35	1383912	NORMAL	NORMAL	NA	NA	NA	NA	9.8	4.3	1.72	0.46	NO GROWTH	REFER	PASS	-
36	1383907	NORMAL	NORMAL	NA	NA	NA	NA	14	3.1	1.49	2.7	NO GROWTH	REFER	REFER	hearing loss
37	1388966	NORMAL	NORMAL	NA	NA	NA	NA	14.2	3.7	2.07	0.44	NO GROWTH	REFER	PASS	-
38	1289844	NORMAL	NORMAL	NA	NA	NA	NA	10.5	15	8.25	2	NO GROWTH	REFER	PASS	-
39	1392246	NORMAL	NORMAL	NA	NA	NA	NA	7.1	6.28	1.58	0.5	NO GROWTH	REFER	PASS	-
40	1267379	NORMAL	NORMAL	NA	147	38	no growth	6.9	12	9.00	0.44	NO GROWTH	REFER	REFER	no hearing loss
41	1483910	NORMAL	NORMAL	2.5	NA	NA	NA	12.9	7.2	3.44	0.8	NO GROWTH	PASS	-	-
42	1387581	NORMAL	NORMAL	1.38	NA	NA	NA	15.8	3.6	1.78	0.23	NO GROWTH	REFER	PASS	-
43	1482320	NORMAL	NORMAL	4.7	NA	NA	NA	14.2	9.2	7.56	4.3	NO GROWTH	REFER	PASS	-
44	1384201	NORMAL	NORMAL	3.19	NA	NA	NA	17	8.2	5.23	0.6	NO GROWTH	PASS	-	-
45	1432210	NORMAL	NORMAL	10.3	50	70	no growth	14.8	7.8	4.12	0.53	NO GROWTH	PASS	-	-
46	1130677	NORMAL	NORMAL	14.8	NA	NA	NA	11.2	5.3	2.30	0.87	NO GROWTH	PASS	-	-
47	1432010	NORMAL	NORMAL	NA	NA	NA	NA	18	2.3	0.98	4.3	NO GROWTH	REFER	REFER	no hearing loss
48	1416320	NORMAL	NORMAL	4.3	NA	NA	NA	14.3	7.8	5.26	0.44	NO GROWTH	PASS	-	-
49	1367133	NORMAL	NORMAL	4.8	NA	NA	NA	15.8	15.8	11.60	0.5	NO GROWTH	REFER	REFER	no hearing loss
50	1389681	NORMAL	NORMAL	29.6	48	85	no growth	9.9	4.5	1.98	5.8	NO GROWTH	REFER	REFER	no hearing loss

N

Baba Farid University
of Health Sciences
Sadiq Road
Faridkot-151 203 (Pb). INDIA.
Phone : 01639-256232,256236
Tele/Fax : 01639-256235
Email Examination@bfuhs.ac.in

No. BFUHS/2K21p-TH/ 414 Date 05/4/22

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Deeksha Chawla**, a student of your college for **MS Otorhinolaryngology (ENT)** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Screening for hearing loss in high risk neonates using otoacoustic emissions.

Supervisor
Dr. Rohit Verma
Assoc. Prof.,
Dept. of E.N.T.,
DMCH, Ludhiana.

Co-Supervisor

Dr. Manish Munjal
Prof. & Head,
Dept. of E.N.T.,
DMCH, Ludhiana.

Dr. Siddharth Bhargava
Assoc. Prof.,
Dept. of Paediatrics,
DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

149. If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.

150. The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.

SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MASTER OF SURGERY

IN

OTORHINOLARYNGOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

MARCH, 2021

DR DEEKSHA CHAWLA

DEPARTMENT OF OTORHINOLARYNGOLOGY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS
For the degree of	M.S. ENT
Name of the candidate	Dr. Deeksha Chawla
Supervisor	Dr. Rohit Verma, Associate Professor, Department of ENT, DMCH, Ludhiana
Co-Supervisor	Dr. Manish Munjal, Professor & Head, Department of ENT, DMCH, Ludhiana Dr. Kamaldeep Arora, Associate Professor, Department of Pediatrics, DMCH Ludhiana
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Otoacoustic Emissions (OAE) are the sounds that are generated from within the inner ear. They are clinically important because they are the basis of a simple, non-invasive test for cochlear hearing loss in high risk neonates.

Aims and Objectives: The primary objective is to estimate the incidence of hearing loss in high risk neonates admitted in neonatal ICU at Dayanand Medical College & Hospital using otoacoustic emissions testing as a screening tool and secondary objective is to determine the risk factors predictive of hearing impairment in neonates.

Materials and Methods: In this prospective observational study, the high risk neonates admitted to NICU, who will qualify the high risk category criteria will be assessed for hearing loss using OAE as a screening tool

Inclusion Criteria: All neonates enrolled in the trial, will have informed parental consent and must have at least one of the risk factors: (in accordance to the guidelines given by Joint Committee on infant hearing) 1. Family history of hereditary childhood sensorineural hearing loss. 2. In utero infections (toxoplasmosis, rubella, cytomegalovirus, herpes simplex virus infections and syphilis) 3. Craniofacial anomalies 4. Low birth weight <2500 g (Indian population) 5. Hyperbilirubinemia at serum levels requiring exchange transfusion 6. Ototoxic medications 7. Bacterial meningitis 8. Perinatal asphyxia (APGAR of 5 at 1 minute or of 6 at 5 minutes) 9. Mechanical ventilation lasting 5 days or longer 10.Stigmata or other findings associated with a syndrome known to include a sensorineural hearing loss.

Exclusion Criteria: 1. Neonates with congenital external ear malformation precluding the use of OAE as a screening tool.2. Neonates with non consenting parents.

Clinical significance: Early detection of hearing loss in high risk neonates and hence their timely rehabilitation with an aim to reduce the rates of morbidities. Screening of high risk neonates for hearing loss.

Keywords: Otoacoustic emissions, High risk neonates, Sensorineural hearing loss, Neonatal hearing loss

(Signature) Candidate	(Signature) Co-supervisor	(Signature) Co-supervisor	(Signature) Supervisor	(Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS"** exist in the Department of ENT at Dayanand Medical College and Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

Dr. Rohit Verma
Associate Professor
Department of ENT
DMCH, Ludhiana

Co-Supervisor

Dr. Manish Munjal
Professor & Head
Department of ENT
DMCH, Ludhiana

Dr. Kamaldeep Arora
Associate Professor
Department of Pediatrics
DMCH, Ludhiana

Head of the Department

Dr. Manish Munjal
Professor & Head
Department of ENT
DMCH, Ludhiana

Place : _____

Date : _____

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Deeksha Chawla
Department	ENT
Topic of Thesis	SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS
Likely date of appearing in PG examination	MAY 2023
Date of enrollment	JULY 25, 2020
Name of Head of Department	Dr. Manish Munjal
1) **Supervisor** 2) **Co-Supervisor(s)**	Dr. Rohit Verma Dr. Manish Munjal Dr. Kamaldeep Arora
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)	Dr. Sandeep Kaushal (Dean Academics & Professor & Head, Pharmacology)
Dr. Gurpreet S Wander (Vice Principal)	Dr. Gurpreet S Wander (Vice Principal & Professor & Head, Cardiology)
	Dr. Gagandeep Singh (Secretary, Ethics Committee Professor & Head, Neurology)

Approved: Yes / No

Dr. Sandeep Puri
Chairperson
Research Committee

Approved: Yes / ~~No~~

Prof. Arvind Malhotra
Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES,

FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR

DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr. Deeksha Chawla 51/C Moti Nagar, Ludhiana
2	NAME OF THE INSTITUTE	Dayanand Medical College and Hospital, Ludhiana
3	COURSE OF STUDY AND SUBJECT	MS ENT
4	DATE OF ADMISSION OF COURSE	25-07-2020
5	TITLE OF THE TOPIC	SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

WHO estimates that the people with hearing loss (defined as loss > 40dB) worldwide, has risen from 120 million in 1995 to at least 278 million in 2005 to around 466 million in 2020[1-3]. In the year 2012, 360 million people in the world with disabling hearing loss (5.3% of the world's population) with 32 million (9%) of these being children[2] with an increase to 34 million children in 2020.

With such high rates of hearing loss, there is an enhancement in the morbidity of the pediatric age group and hence causing the prolonged disability. If not detected at an early stage, it further hampers speech, cognition and language. Thus causing delayed speech and language and poor academic performance which can be devastating for the child. Such an increasing trend in the hearing loss has led to the need to screen at an early stage where the consequences are reversible. There is some evidence that early rehabilitation is effective in reducing this impairment[4].

Infact it is a well proven fact that if hearing loss is identified and remediated at an early stage (< 6 months) and provided with corrective intervention (<1 year), the cognitive skills and language can be developed[5].

As stated by the American Academy of Pediatrics, in a published 'position statement' in 1982, the screening of high risk infants should start by the age of 3 months and rehabilitation to commence by 6 months of age[6].

The aim of a newborn hearing screening programme should be to detect severe to profound hearing losses and to reduce the age at which intervention is begun[7]. The National Institute of Health (NIH) gave a consensus statement in 1993 in which it recommended that the universal newborn hearing screening should be conducted by the age of 3 months and also stated that otoacoustic emission might be the technology used for screening[8].

Any screening test that is used should be a feasible test which can be performed without professional training. It should preferably be a

bedside test that does not require shifting the patient to specialised laboratory setups.

There are a combination of subjective and objective testing modalities for hearing loss. Subjective tests include pure tone audiometry, speech audiometry and reflex audiometry. These tests are not practical in newborns as subject's active participation is required. Objective tests include laboratory tests and bedside tests. For sensorineural hearing loss, tests like Otoacoustic Emission testing (OAE), Brain Stem Evoked Response (BERA), [also known as auditory brainstem response (ABR)], and Auditory Steady State Response (ASSR) can be used whereas for conductive hearing loss, Impedence audiometry is the objective test of choice.

OAE and BERA are the two feasible tests that can be conducted to assess sensorineural hearing loss but OAE can be preferred over BERA as BERA involves shifting the baby to specialised laboratory facilities while OAE involves bedside screening of babies. Hence OAE is one such intervention that can be used for early detection of hearing loss in newborns.

Otoacoustic emissions (OAE) are the low intensity sounds produced by the outer hair cells of a normal cochlea and can be elicited by a very sensitive microphone placed in the external ear canal and analysed by the computer[9]. The direction of the flow of the sound is reverse, traveling from the Outer hair cells → Basilar Membrane→Perilymph →Oval Window→Ossicles→Tympanic Membrane→Ear Canal. The presence of otoacoustic emissions is indicative of a healthy ear and its absence that of the damaged ear and thus aiding in the assessment of cochlear function. Otoacoustic emission testing is also an efficient tool to differentiate between cochlear and retrocochlear pathologies. Types of OAE are:

1. Spontaneous OAE which are present in healthy normal hearing people where hearing loss does not exceed 30dB.

2. Evoked OAE which is further of two types:

A) Transient evoked OAE (TEOAE) which is evoked by clicks. In this a series of click stimuli are presented at 80-85dB.

B) Distortion product OAE (DPOAE) In this two tones are simultaneously presented to the cochlea to produce distortion products. They have been used to test hearing in the range of 1000-8000 Hz.

This study will primarily focus on the high risk neonates and hence will screen the neonates for the occurrence of hearing loss and its timely detection. It will also provide an early intervention to rehabilitate the high risk neonates. With otoacoustic emission testing (OAE) as a screening tool, patients admitted in Neonatal ICU, who will qualify the high risk category criteria, will be screened.

With the screening facility available to the high risk neonate at the bedside, the rates of identification of the hearing loss, if any, can be hastened and the morbidities can be dismantled at an earlier stage. Such a study has not been conducted in our region and hence screening the neonates at hospital levels at an early phase of life[10] would help to do a timely intervention, if required.

6.2 REVIEW OF LITERATURE

Epidemiology:

According to the recent estimates, permanent hearing loss of greater than 25 decibel hearing level in poorer ear is present in atleast 4 (1.1 to 6) per 1000[11] As many as half of these infants have no risk factors for hearing loss; thus hearing loss may not be suspected until the child misses language milestones. The prevalence of hearing loss in high risk infants is estimated to be between 2.5 and 10%[12,13]. Universal screening of newborns for the detection of hearing loss aims to detect hearing impairment including unilateral or bilateral sensory or conductive hearing loss averaging 30 – 40 decibel or more in frequency region 500 to 4000 HZ. Hearing impairment in this range will have most impact on speech acquisition[8] In India, incidence of hearing impairment in neonates (at risk and not at risk) ranges from 6 – 60 per 1000 neonates with an average of 4 per 1000 neonates[14] Another survey in India has shown 4 out of every 1000 infants born were found to have severe hearing loss[15].

An otoacoustic emission (OAE) is a sound that is generated from within the inner ear. Having been predicted by Austrian astrophysicist Thomas Gold in 1948, its existence was first demonstrated experimentally by British physicist David Kemp in 1978,[16] The eventual discernment of OAEs in normal ears by Thomas Gold[17] propelled towards the need to look for the hair cell motility[18]. Outer hair cell electromotility is a rapid, force generating, change in the length in response to electrical stimulation[19]. DC electrical pulses either elongate or shorten the outer hair cell and sinusoidal electrical stimulation results in mechanical oscillations at acoustic frequencies. The mechanism underlying outer hair cell electromotility is thought to be the origin of spontaneous otoacoustic emissions. This stimulated response has enlightened the path towards the analysis of preneural cochlear function in the normal ear, quickly without electrodes, using ear canal acoustics.

Brainstem evoked response audiometry (BERA) screening for auditory function is commonly applied to newborn at an increasing risk of

hearing impairment. It is also known as auditory brainstem response (ABR). Implementing such a test programme with BERA requires: considerable resources and organisation. It is a highly time consuming test, taking 30 minutes or more per child. It requires expertise and a skilled operator to obtain and interpret the ABR data and shifting patients to specialised laboratory facilities.

Otoacoustic emission testing on the other hand has long been recognised as potentially a faster method of screening than BERA which requires less expertise and resources to function and is a bedside testing.

Stevens JC et al[20] proposed that the efficiency of neonatal screening could be improved by using OAE testing as the primary screen, with failure referred for ABR screening.

Bhatt J et al[21] compared the sensitivity and specificity of OAE and BERA in neonatal screening and concluded, : The incidence of permanent congenital hearing loss according to diagnostic testing was five out of hundred newborn with risk factors (5%). And in well nursing babies was 0.5% which is 10 times less than the high risk population. Sensitivity & specificity of OAE was 70% and 61% at 0 months and 70% and 99% at 3 month and for BERA sensitivity and specificity at 3 months was 90% and 99% respectively and at 6 months 100% and 99% respectively.

Kennedy CR et al[22], in their study compared the use of ABR and OAE as a screening tool in high risk infants and concluded that automated OAE testing of both ears was quickest (median 5-12 minutes) and least invasive (no scalp electrodes). Bilateral failure rates (and upper 95% confidence limits) with a stimulus 35-36 dB above normal hearing threshold level (nHL) were 3 0% (4.6) with automated OAE, 3-2% (5.1) with ABR, and 2-7% (4.4) with automated ABR. Automated OAE was the test most sensitive for subsequently confirmed hearing impairment.

Kemp DC et al[23] in their study used otoacoustic emissions as an objective neonatal auditory screener and studied the role of otoacoustic emissions in early detection programmes, and illustrated the present status of efforts to evolve reliable screening techniques. They felt that

the target for OAE usage should be neonatal screening on a routine basis in the maternity unit, on a much wider population than is currently considered feasible with ABR methods.

Nagapoornima P et al[14] conducted a prospective non-randomised study to determine the incidence of hearing impairment in at risk and not at risk neonates in tertiary care hospital in Bangalore.The incidence of hearing impairment was 7 out of 1490 in not at risk newborns and 3 out of 279 in at risk newborns.

Meyer C et al[24] conducted a study to determine the incidence and risk factors for hearing disorders in a selected group of neonates and the feasibility of selective hearing screening. and summarised the risk factors for neonatal hearing disorders. Craniofacial malformations, familial hearing disorders, and neonatal bacterial infections were significant factors associated with pathologic A-ABR (Automated ABR) testing. In contrast, very low birth weight and complications of prematurity were not independent risk factors for pathologic screening results in their study population.

Heinemann M et al[25] conducted a study to find the cost effectiveness of newborn hearing with different instruments and found that two step screening(first with OAE and then with BERA) was most cost effective.

Anand S et al[26] conducted a prospective study on neonatal hearing screening. In this study they screened 1052 newborns with a two staged OAE and those who failed the two staged OAE were referred to the brain stem evoked response (BERA) testing. He concluded, OAE can be used as a neonatal screening tool as well as for the evaluation of a suspected pediatric population to help diagnose childhood hearing impairment at a very early stage, allowing for the rehabilitation of the child within the critical period for speech and language development.

Khairi MM et al [27] conducted a study on hearing assessment. 401 at risk neonates were included in the two stage study and it was concluded that mechanical ventilation of more than 5 days was not an independent risk factor for hearing impairment.

Bansal S et al [28] conducted a study on role of transient evoked otoacoustic emissions in hearing screening programs in developing

countries. The objective of this program was to formulate a protocol for infant hearing screening in developing countries enabling it to be later incorporated into their national deafness screening programs. 2659 infants in the age range of 0-3 months were included in study. They were divided into 3 groups with age range between 0-1, 1-2 and 2-3 months of age. All were subjected to transient evoked otoacoustic emission (TEOAE) for hearing screening. Those who failed first screening were followed up after 1-month. Infants who had failed the second screening underwent Brainstem Evoked Response Audiometry (BERA). This study highlighted that delayed hearing screening at 3 months of age would considerably decrease the number of false positive cases.

Sharma P et al [29] conducted a study to evaluate BERA as an objective test of hearing assessment in icteric babies and concluded that BERA is a simple, reliable and effective technique for detection of hearing impairment in the neonates.

6.3 AIMS AND OBJECTIVES OF THE STUDY

1. PRIMARY OBJECTIVE: To estimate the incidence of hearing loss in high risk neonates admitted in neonatal ICU at Dayanand Medical College & Hospital using otoacoustic emissions testing as a screening tool.
2. SECONDARY OBJECTIVE: To determine the risk factors predictive of hearing impairment in neonates.

7. MATERIALS AND METHODS

7.1 Source of Data

It is a Prospective Observational Study that will be conducted for duration of 1 year from 1st April 2021 to 31st March 2022 in High risk Neonates admitted in Neonatal ICU of Dayanand Medical College and Hospital, Ludhiana.

OTOACOUSTIC EMISSIONS TEST:

It is an objective test. It is a bedside test and takes about 1-5 minutes. It can be performed in a sleeping or awake neonate. The probe is to be kept in the external auditory canal. The OAE emitted from the cochlea are detected using the probe. The interpretation of the test is in the form of PASS or REFER. PASS is suggestive of a normal ear and REFER may either indicate an abnormal ear or a false positive test.

HIGH RISK NEONATE: A high risk neonate is defined as any neonate with a gestational age of less than 34 weeks or with gestational age of more than 34 weeks but with co morbidities like birth weight of < 2500g , hyperbilirubinemia , bacterial meningitis , perinatal asphyxia , requiring mechanical ventilation and others that hamper the normal course of life of the neonate.

7.2 METHOD OF COLLECTION OF DATA

It will be a prospective observational study. The high risk neonates will be screened for hearing loss using otoacoustic emissions (OAE) in two stages. In OAE stage 1 high risk neonates admitted in NICU will undergo otoacoustic emission test prior to discharge. The interpretation of the test will be in the form of REFER or PASS. The neonates in whom OAE results are ‘REFER’ will undergo a repeat OAE after 6 weeks (Stage 2 OAE) .Those neonates in whom results are ‘PASS’ will be considered with normal hearing and will not be followed up. Neonates in whom results are still ‘REFER’ will undergo BERA test to determine the degree of hearing loss. Counseling of parents regarding the rehabilitation of hearing shall be undertaken of those neonates in whom OAE is reported as REFER and BERA shows hearing loss.

INCLUSION CRITERIA

All neonates enrolled in the trial, if informed parental consent is obtained and atleast one of the risk factors according to the Joint Committee on Infant Hearing is found:

1. Family history of hereditary childhood sensorineural hearing loss
2. In utero infections (toxoplasmosis, rubella, cytomegalovirus, herpes simplex virus infections, and syphilis)
3. Craniofacial anomalies
4. Low birth weight <2500 g (Indian Population)
5. Hyperbilirubinemia at serum levels requiring exchange transfusion
6. Ototoxic medications
7. Bacterial meningitis
8. Perinatal asphyxia (APGAR of 5 at 1 minute or of 6 at 5 minutes)
9. Mechanical ventilation lasting 5 days or longer
10. Stigmata or other findings associated with a syndrome known to include a sensorineural hearing loss

EXCLUSION CRITERIA

1. Neonates with congenital external ear malformation precluding the use of OAE as a screening tool.
2. Neonates with non consenting parents.

STUDY DURATION:

1 year from 1st April 2021 to 31st March 2022

OUTCOME: Screening of high risk neonates for hearing loss.

STATISTICAL ANALYSIS

Data will be described in terms of range, mean±standard deviation (±SD), frequencies (No of cases) and relative frequencies (%age) as appropriate.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Yes, this study will require high risk neonates on whom otoacoustic emissions will be used as a screening tool for hearing loss assessment.

A consent will be taken from the neonate's parents before conducting any intervention.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached herewith

LIST OF REFERENCES

1. World Health Organization. Primary ear and hearing care training resource. World Health Organization; 2006.
2. W WHO M. WHO global estimates on prevalence of hearing loss. Mortality and Burden of Diseases and Prevention of Blindness and Deafness WHO; 2012.
3. Resnikoff S, Pascolini D, Etya'ale D, Kocur I, Pararajasegaram R, Pokharel GP, et al. Global data on visual impairment in the year 2002. Bull World Health Organ. 2004;82:844-51.
4. Markides A. Shortpaper: Age at fitting of hearing aids and speech intelligibility. British journal of audiology. 1986;20:165-7.
5. World Health Organization. Newborn and infant hearing screening: Current issues and guiding principles for action. Geneva: WHO; 2010.
6. American Academy of Pediatrics. Position statement 1982-Joint Committee on Infant Hearing. Pediatrics. 1982;70:7.
7. Sancho J, Hughes E, Davis A, Haggard MP. Epidemiology of hearing impairments in children and its application to screening. Pediatric Audiology 0-5 Years. Taylor and Francis, London; 1988. p. 1-35.
8. Universal hearing screening in Illinois 2003-2004, published report of Illinois Dept of Public health, Division of Epidemiologic studies, August; 2006. p. 1-11.
9. Lilaonitkul W, Guinan Jr JJ. Reflex control of the human inner ear: a half-octave offset in medial efferent feedback that is consistent with an efferent role in the control of masking. Journal of neurophysiology. 2009;101:1394-406.
10. Kemp DT, Ryan S, Bray P. A guide to the effective use of otoacoustic emissions. Ear and hearing. 1990;11:93-105.
11. White KR, Maxon AB. Universal screening for infant hearing impairment: simple, beneficial, and presently justified. International journal of pediatric otorhinolaryngology. 1995;32:201-10.

12. Abdullah A, Hazim MY, Almyzan A, Jamilah AG, Roslin S, Ann MT, et al. Newborn hearing screening: experience in a Malaysian hospital. Singapore medical journal. 2006;47:60.
13. Asma A, Wan Fazlina WH, Almyzan A, Han YS, Jamilah AG, Roslin S. Benefit and pitfalls of newborn hearing screening. Med J Malaysia. 2008;63:293-7.
14. Nagapoornima P, Ramesh A, Rao S, Patricia PL, Gore M, Dominic M, et al. Universal hearing screening. Indian J Pediatr. 2007;74:545-9.
15. Ansari MS. Screening programme for hearing impairment in newborns: A challenge during rehabilitation for all. Asia pacific disability rehabilitation journal. 2004;15:83-9.
16. Kemp DT. Stimulated acoustic emissions from within the human auditory system. The Journal of the Acoustical Society of America. 1978;64:1386-91.
17. Gold T. Historical background to the proposal 40 years ago, of an active model for cochlear frequency analysis. In Cochlea mechanisms: structure, function and models. New York: Plenum; 1989. p. 299-305.
18. Kemp DT, Ryan S. Otoacoustic emission tests in neonatal screening programmes. Acta Oto-Laryngologica. 1991;111:73-84.
19. Goutman JD, Fuchs PA, Glowatzki E. Facilitating efferent inhibition of inner hair cells in the cochlea of the neonatal rat. The Journal of physiology. 2005;566:49-59.
20. Stevens JC, Webb HD, Hutchinson J, Connell J, Smith MF, Buffin JT. Click evoked otoacoustic emissions in neonatal screening. Ear and hearing. 1990;11:128-33.
21. Bhatt J, Chhangte L. Accuracy of OAE and BERA to Detect the Incidence of Hearing Loss in Newborn. International Journal of Scientific and Research Publications; 2015. p. 1-6.
22. Kennedy CR, Kimm L, Dees DC, Evans PI, Hunter M, Lenton S, et al. Otoacoustic emissions and auditory brainstem responses in the newborn. Arch Dis Child. 1991;66:1124-9.

23. Kemp DT, Ryan S. Otoacoustic emission tests in neonatal screening programmes. Acta Otolaryngol Suppl. 1991;482:73-84.
24. Meyer C, Witte J, Hildmann A, Hennecke KH, Schunck KU, Maul K, et al. Neonatal screening for hearing disorders in infants at risk: incidence, risk factors, and follow-up. Pediatrics. 1999;104:900-4.
25. Heinemann M, Bohnert A. Hearing screening in newborn infants. Comparative studies and cost analysis with different instruments. Laryngo-rhino-otologie. 2000;79:453-8.
26. Anand S, Tiwari A, Goyal S. Prospective study for newborn hearing screening-A experience from tertiary care centre in central India. Int J Pediatr Res.2016;3:668-71.
27. Khairi MM, Suhaimi SD, Shahid H, Normastura A. Hearing screening of infants in Neonatal Unit, Hospital Universiti Sains Malaysia using transient evoked otoacoustic emissions. The Journal of laryngology and otology. 2005;119:678.
28. Bansal S, Gupta A, Nagarkar A. Transient evoked otoacoustic emissions in hearing screening programs—Protocol for developing countries. International journal of pediatric otorhinolaryngology. 2008;72:1059-63.
29. Sharma P, Chhangani NP, Meena KR, Jora R, Sharma N, Gupta BD. Brainstem evoked response audiometry (BAER) in neonates with hyperbilirubinemia. Indian J Pediatr. 2006;73:413-6.

PROFORMA

I. I) IDENTIFICATION DATA

1.Name SERIAL NO.

2.C.R. No.

3.Age / Gender:

4.Age of parents:

5.Parental occupation:

6.Address :

State:

City:

Town/Village: Urban/Rural:

7.Birth Order:

8.APGAR score :

II. II) CHIEF COMPLAINTS DURATION

A) ANTENATAL HISTORY

1. Antenatal Problems
   - a)H/o Hypertension
   - b)H/o Preeclampsia
   - c)H/o Diabetes
   - d)H/o Thyroid
   - e)H/o Epilepsy
   - f) H/oSensorineural hearing loss
2. Maternal drug history
   - a) Aminoglycosides
   - b) Diuretics
   - c) Anti epileptics
   - d) Radiation exposure
   - e) Antimalarial drugs
   - f) Any other drugs

Signature of Candidate

Signature of Supervisor

B) BIRTH HISTORY

1) Place of Delivery :
2) Type of Delivery :
3) Asphyxia/Other problems :
4) Birth Weight :
5) Gestation :

C) HIGH RISK FACTOR:

1. Family history of hearing loss	YES/NO
2. TORCH Infection	YES/NO
3. Craniofacial Anomaly	YES/NO
4. Low birth weight < 2.5 kg	YES/NO
5. Hyperbilirubinemia	YES/NO
6. Ototoxic Medications	YES/ NO
7. Bacterial Meningitis	YES/NO
8. Postnatal Asphyxia	YES/NO
9. On Mechanical Ventilation for > 5 days	YES/NO
10. Any stigmata or syndrome associated with hearing loss	YES/NO

III) GENERAL PHYSICAL EXAMINATION

1.Pallor
2.Edema
3.Vitals

IV) LOCAL EXAMINATION

EAR:	RIGHT EAR	LEFT EAR
1.Pinna		
2.External Auditory Canal		
3.Post Auricular area		
4.Pre Auricular Area		
5.Facial Nerve		

Signature of Candidate **Signature of Supervisor**

NOSE ;

THROAT:

NECK:

V) INVESTIGATIONS

1. Serum Bilirubin :
2. CSF:

PROTEIN	GLUCOSE	GRAM STAIN/ CULTURE SENSITIVITY

3.Hb : 4.TLC : 5.ANC

6.CRP: 7. Blood culture

TESTING

A) OAE Testing: OAE findings:

EAR	RIGHT EAR	LEFT EAR
FIRST SCREENING (F2,Hz / F1,dB/ F2,dB/ DP, dB/Noise, dB/ SNR, dB)	REFER / PASS	REFER / PASS
SECOND SCREENING (F2,Hz / F1,dB/ F2,dB/ DP, dB/Noise, dB/ SNR, dB)	REFER / PASS	REFER / PASS

Signature of Candidate

Signature of Supervisor

B) BERA YES/NO

WAVE	IN MILLI SECONDS(ms)
Wave I - III IPL	
Wave III -V IPL	
Wave I - V IPL	
Interaural Latency	
Morphology of Wave V	

RESULT:

PROVISIONAL DIAGNOSIS:

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o..... am willing to participate in the study **"SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that the intervention test being conducted is for screening of the neonate and no extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"SCREENING FOR HEARING LOSS IN HIGH RISK NEONATES USING OTOACOUSTIC EMISSIONS" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ

_____ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਮਿਤੀ_____

ਪਤਾ _____

ਪਤਾ _____

_____ ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____ ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ